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Anions

Conversion Table: Milligrams per hundred milliliters to milliequivalents per liter

CHLORIDE	
mg./100 ml.	*mEq./L.*
240	67
260	73
280	79
300	84
320	90
340	96
360	101
380	107
400	113
420	118
440	124

To convert mg./100 ml. to mEq./L. multiply by 0.282

CHLORIDE AS SODIUM CHLORIDE	
mg./100 ml.	*mEq./L.*
430	73
460	78
490	84
520	89
550	94
580	99
610	104
640	109
670	114
700	120
730	125

To convert mg./100 ml. to mEq./L. multiply by 0.171

PHOSPHORUS	
mg./100 ml.	*mEq./L.*
1	0.58
2	1.16
3	1.74
4	2.32
5	2.90
6	3.48
7	4.06
8	4.64
9	5.22
10	5.80

To convert mg./100 ml. to mEq./L. multiply by 0.580

BICARBONATE	
CO_2 Content or CO_2–Combining Power Volumes per 100 ml.	*mEq./L.*
10	4.5
20	9.0
30	13.5
40	18.0
50	22.5
60	27.0
70	31.5
80	36.0
90	40.5
100	45.0
110	49.5

To convert volumes per 100 ml. to mEq./L. multiply by 0.45

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Conversion Table: Milligrams per hundred milliliters to milliequivalents per liter

SODIUM	
mg./100 ml.	*mEq./L.*
270	117
280	122
290	126
300	130
310	135
320	139
330	143
340	148
350	152
360	156
370	160

To convert mg./100 ml. to mEq./L. multiply by 0.435

POTASSIUM	
mg./100 ml.	*mEq./L.*
5	1.28
10	2.56
15	3.84
20	5.12
25	6.40
30	7.68
35	8.96
40	10.24

To convert mg./100 ml. to mEq./L. multiply by 0.256

CALCIUM	
mg./100 ml.	*mEq./L.*
4	2.0
5	2.5
6	3.0
7	3.5
8	4.0
9	4.5
10	5.0
11	5.5
12	6.0
13	6.5
14	7.0

To convert mg./100 ml. to mEq./L. multiply by 0.5

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FLUID BALANCE HANDBOOK FOR PRACTITIONERS

FLUID BALANCE HANDBOOK FOR PRACTITIONERS

By

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To

WILLIAM DANIEL SNIVELY, SR., M.D.

a practitioner of medicine

for 42 years

Preface

The authors' goal in preparing this handbook has been to provide the practicing physician with an inviting, clear and usable text on *clinical* fluid balance.

Various steps have been taken to insure the achievement of this goal. First of all, we have made free and unabashed use both of memory helps and of visual aids. William James said: *The more other facts a fact is associated with in the mind, the better possession of it our memory retains. Each of its associates becomes a hook to which it hangs, a means to fish it up by when sunk beneath the surface.* Aids such as we have used are not required for the investigator. Suffice it to say, they have proved immensely useful in teaching this subject to practitioners.

In our journey through the land of fluid balance, we have kept on the main highways, avoiding excursions down the byways of theory, unnecessary detail, controversy and speculation. We have spoken the language of the clinician, not of the biochemist. We have endeavored to view the subject from the standpoint of the needs of the practicing physician. The book is written for him! Hence, our intense effort to include in the book every fact needed by the clinician for the practical diagnosis and treatment of most patients with clinical fluid imbalances.

We like to think of this book as presenting the *minimal basic facts.* We hope it will serve as a point of departure for those physicians who wish to have a broader background in this fascinating subject. To this end we have given much thought to the "References for Additional Reading." There will be found the complete bibliographic reference for any book or article mentioned in the text.

Various facets of our approach to fluid balance have been presented in the *Journal of the Indiana State Medical Association*, *GP*, *The Journal of the Student American Medical Association,* and the *Journal of Pediatrics* and as scientific exhibits at the annual meetings of the American Academy of Pediatrics, American Academy of General Practice, American Medical Association, the American College of Surgeons and the Indiana State Medical Association.

Important in the achievement of our goal has been the artistry of Kathleen Calhoun. Of great help have been the counsel and production skills of Charles C Thomas, Publisher, whose encouragement and cooperation have been unflagging at every juncture. Our literary approach to the subject was influenced by that charming monograph on oceanography *The Sea Around Us* by Rachel Carson.

In order to insure the clarity and usefulness of the book, we submitted the preliminary manuscript to a panel of some 40 practitioners, academic physicians and technically trained laymen. Their helpful suggestions and discerning criticism have contributed much to the handbook. It is a pleasure to express our gratitude to these co-workers.

Obviously, this handbook presents neither new concepts nor results of original investigations. In its attempt to make available to the practicing physician the many advances in the field of fluid therapy, it depends heavily on the work of outstanding basic and clinical investigators, the list of which is too long to reproduce here. Nevertheless, special mention should be made of Allan M. Butler, Daniel C. Darrow, Robert Elman, James L. Gamble, Charles U. Lowe, Carl A. Moyer, Nathan B. Talbot and Harry F. Weisberg, whose basic investigations and extensive writings have helped immeasurably in the preparation of this text.

Our sincere appreciation is extended to Phyllis Moffett who typed the manuscript, and to Martha Wessner, Eino J. Macklin, Orville P. Nuffer and Howard D. Evans for valuable suggestions and counsel. We would like to thank D. Mead Johnson, President of Mead Johnson & Company, for his interest and encouragement.

The authors, members of the Medical Department of Mead Johnson & Company, prepared this handbook as an independent project. The views expressed in it are the authors' and do not necessarily coincide with the official views of Mead Johnson & Company on the various aspects of fluid balance.

It is the chief desire of the authors that this primer will orient the reader with respect to the clinical disturbances of the body fluids and will stimulate him to the intensive study required for practical mastery.

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FLUID BALANCE HANDBOOK FOR PRACTITIONERS

Part I Foundation Facts

1

First Things First

Don't place the cart before the horse!
OLD MAXIM

SINCE that not-too-remote period of history when the art of medicine emerged from the miasmal swamps of witchcraft, magic and superstition, the approach to the diagnosis and management of disease has been chiefly clinical. Our introduction, as medical students, to such pathologic states as rheumatic fever, appendicitis, diphtheria and lobar pneumonia was to "clinical pictures." These included the clinical causes of the diseases and the clinical findings, both subjective and objective. Clinical history and physical examination were presented as paramount. Such matters as pathology, pathologic physiology and laboratory findings were regarded as of real, though perhaps secondary, import.

The clinical approach loses none of its validity when one undertakes the study of the various clinical fluid imbalances. Bear in mind that fluid imbalances are not mere biochemical appendages grafted onto disease states; rather, they are clinical entities in themselves, with their own clinical causes, clinical findings, laboratory findings and therapy. Like other clinical entities, they modify and are modified by coexisting pathologic states.

The fact that imbalances are frequently mixed does not relieve the clinician of the necessity of recognizing the individual clinical pictures of specific imbalances any more than it relieves medical students of the necessity of learning the individual clinical pictures of pulmonary tuberculosis, gastric carcinoma, or acute hepatitis. Such clinical entities, like those of fluid imbalance, may be confusingly intermingled, as Jaffé so frequently emphasized.

For that great group of practitioners who have few or no laboratory facilities available, familiarity with the clinical approach is especially valuable. It is also helpful to those who, because of unavailability of emergency 24-hour laboratory service, must assess a patient, make a diagnosis and proceed with therapy while awaiting a laboratory report. Help from the laboratory is often extremely valuable and is not to be decried; nonetheless, it should be relegated to its rightful position.

Just as clinical considerations are of primary importance in diagnosis, so should they be foremost in therapy. Certainly clinical management of the patient deserves precedence over management of his biochemical findings. Edwards Park tells of the Sister whose grief at the death of an infant with diarrhea was eased by the fact that before the baby died, he had had a "beautiful stool." Similarly, the correction of an abnormal plasma chemical level is scant satisfaction to the patient if he is not alive to enjoy it.

To view the clinical picture as of primary importance, to regard laboratory findings as confirmatory rather than diagnostic, to treat patients rather than plasma chemical levels is to introduce no new concept. Rather, it is to adhere to an old and respected practice, to put the clinical Dobbin before the biochemical cart, which is where he belongs.

In an article titled "Contributions of the Laboratory to the Practical Management of Disorders of Body Water and Electrolyte," which all should read, Robert E. Cooke has put it rather well: "Regardless of the multiplicity of laboratory determinations, proper interpretation and therapy depend nonetheless upon frequent and accurate clinical evaluation."

Before proceeding with the intriguing matter of the several fluid imbalance entities, it is essential that we review a few "foundation facts." Only those required for a working knowledge of fluid balance will be covered. . . .

2

An Ancient Fluid

. . . before our extremely remote ancestors could come ashore to enjoy their Eocine Eden or their Paleozoic Palm Beach, it was necessary for them to establish an enclosed aqueous medium which would carry on the role of sea water . . .

James L. Gamble

Human extracellular fluid, that salty liquid which bathes our body cells, is truly an ancient fluid. Its primordial ancestor was ocean water of the Paleozoic seas of a third of a billion years ago. It was during this period that the remote ancestors of the human race forsook their marine homeland and ventured ashore to populate the greening continents.

Simple marine organisms require a salty fluid environment for the functioning of the myriads of chemical processes necessary for life. When these creatures made their first landfall they, perforce, brought a parcel of their oceanic environment with them, as their body extracellular fluid. In man, as in simpler forms of life, the extracellular fluid serves both as a transport system and as a great chemical reaction vat in which untold numbers of reactions of almost unbelievable complexity are proceeding at all times.

The chemical composition of extracellular fluid has so faithfully been preserved over the ages that the extracellular fluid of human beings resembles, both in its concentration and in its composition, the waters of that prehistoric day when life made its first landfall.

The body cells contain their own fluid—the cellular fluid—as opposed to the extracellular fluid which surrounds the cells. We prefer the term "cellular" to the more frequently used "intracellular" in referring to the fluid within the cells. "Cellular" is just as meaningful and has the advantage of brevity. The term "body fluid" includes both the cellular and the extracellular fluid.

SUMMARY

Sea water of an ancient geologic era is the phylogenetic ancestor of human extracellular fluid, the salty liquid which surrounds the body cells.

3

Body Fluid Types

. . . Blood is thicker, sir, than water, now as then . . .
WALTER RICE

WATER, the universal solvent of chemistry, also serves this function in the human body. The body fluids, however, consist not only of water but of important dissolved materials. The portion of these dissolved materials which is important from the standpoint of clinical fluid balance includes those "dynamic chemicals" of the body fluids, the electrolytes.

The body fluids are properly divided into fluid contained within the body cells—the *cellular fluid*—and the fluid which surrounds and bathes the cells—the *extracellular fluid.* The cellular fluid and the extracellular fluid comprise the *body fluids.* About 60% of the weight of the adult body consists of fluids with the remaining 40% solids. The fluid inside the cells—the cellular fluid—makes up 45% of the body weight, and the fluid outside the cells—the extracellular fluid—makes up 15% (see Figure 1).

In the case of the newborn infant, approximately 77% of the body weight consists of body fluids with only 23% solids. The infant's cellular fluid makes up 48% of his body by weight and his extracellular fluid 29% (see Figure 2). Thus, it is seen that the young infant has nearly twice as much extracellular fluid as the adult in relation to his size. Perhaps the infant's relatively large extracellular fluid volume is rendered necessary by his greater fluid turnover.

One reason why both the infant and the child lose fluid more rapidly than the adult is the fact that they are rapidly growing individuals with high rates of metabolic activity. A second reason

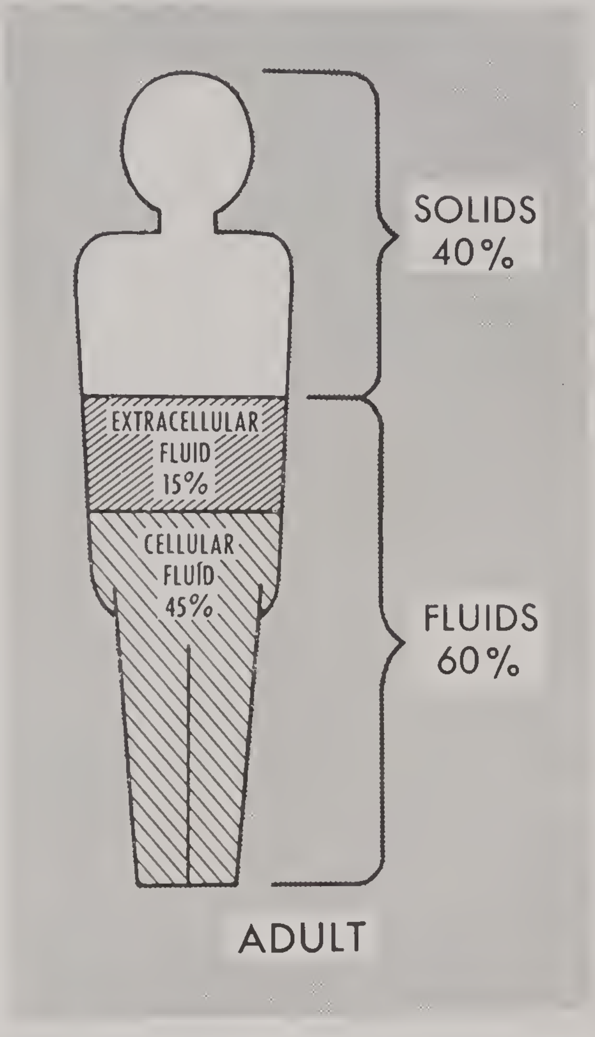


Figure 1. Fluid and Solid Components of Body Weight in the Adult.

is the fact that the skin area of the infant is two to three times that of an adult in proportion to its volume of body fluid. Recall that the skin and lungs in both the infant and the adult account for about one-half of the daily loss of water in health, and even more in certain disease states.

Let us visualize the infant and the adult as two garden sprinkling cans. The size of the can proper corresponds to body fluid volume and the area of the sprinkling spout to skin surface. The infant with its relatively smaller body fluid volume, corresponding to the can proper, will more rapidly deplete its body fluid through its large skin surface, corresponding to the sprinkling spout, than will the adult (see Figure 3).

Because of these factors, the infant without any water intake

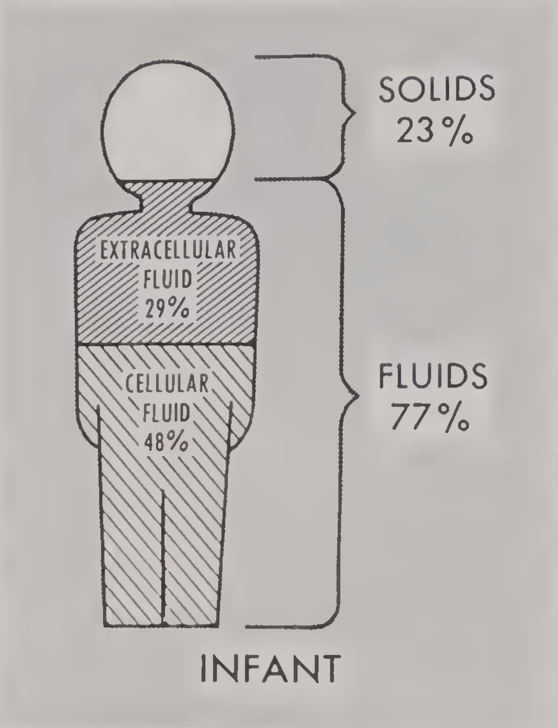


Figure 2. Fluid and Solid Components of Body Weight in the Infant.

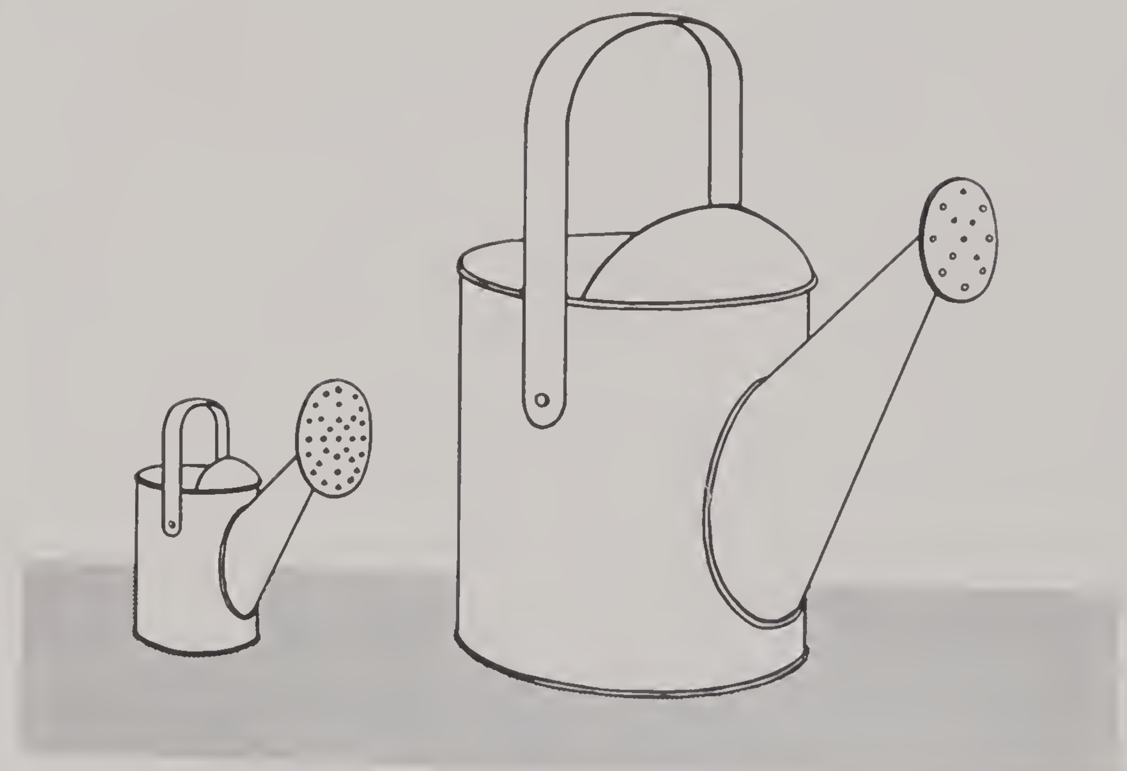


Figure 3. The Sprinkling Can Analogy.

will lose a volume of fluid equal to its extracellular fluid in 5 days, while it will take the adult about 10 days to lose a volume of fluid equal to his extracellular fluid. It appears that the relatively large volume of extracellular fluid in the infant may be designed to compensate for his greater vulnerability to fluid loss.

As the infant grows the relative amounts of cellular and extracellular fluid progress toward the adult values. Indeed, chemical maturity is reached quite early, at about the age of 2½ years.

The extracellular fluid can be divided into two general types on the basis of its location. Extracellular fluid inside the blood vessels is designated the *intravascular* extracellular fluid, or the *plasma.* Extracellular fluid lying outside the blood vessels is called the *extravascular* extracellular fluid, or the *interstitial fluid.* We have no precise knowledge concerning the differences in composition of the fluids within the various cells of the body, so at the present time we do not subdivide the cellular fluid.

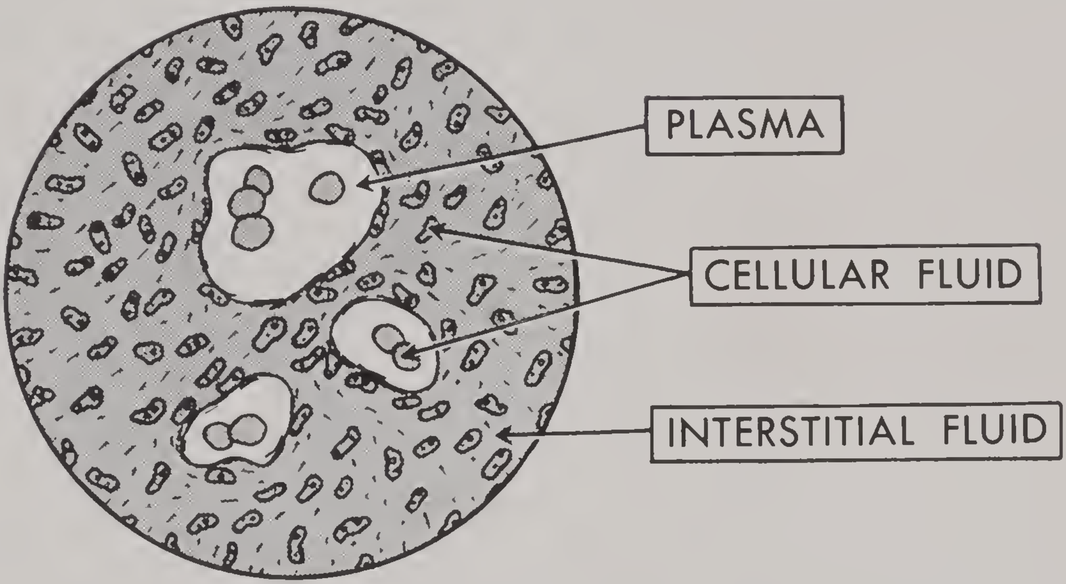


Figure 4. Microscopic Anatomy of Body Fluids.

The spaces which the body fluids occupy are sometimes referred to as "compartments." While the spaces containing the plasma and the interstitial fluid might be regarded as compartments since each is continuous, the term "compartment" is misleading when applied to the space containing the cellular fluid. This space is divided into billions of tiny compartments, each bounded by a cellular membrane (see Figure 4).

SUMMARY

1) In addition to the cellular fluid, all verterbrates possess extracellular fluid.

2) The extracellular fluid and the cellular fluid comprise the body fluids.

3) About 60% of the weight of the adult body consists of fluids, while 40% is solids.

4) The cellular fluid constitutes 45% of the adult body by weight, and the extracellular fluid 15%.

5) About 77% of the weight of the newborn body is fluids, while 23% is solids.

6) The newborn infant's cellular fluid constitutes 48% of his body by weight and his extracellular fluid 29%.

7) The body fluids of the infant and the child are far more rapidly depleted than are those of the adult.

8) There are two types of extracellular fluid: extracellular fluid inside the blood vessels, called *intravascular* extracellular fluid or *plasma;* and extracellular fluid outside the blood vessels, called *extravascular* extracellular fluid or *interstitial fluid.*

9) The cellular fluid is not subdivided.

4

Units of Measure

> *. . . if they should make the standard for the measure we call a "foot" a Chancellor's foot; what an uncertain measure would this be! One Chancellor has a long foot, another a short foot, a third an indifferent foot . . .*
>
> John Selden

ALL THREE basic types of body fluid—the plasma, the interstitial fluid and the cellular fluid—consist of water and important dissolved substances. Of these substances, the foremost are those active, dynamic chemicals which we designate as "electrolytes." Electrolytes are substances which become ionized, that is, develop electric charges, when placed in water. Those that develop positive charges are known as "cations." An example is sodium (Na^+). If these electrolytes are placed in a wet electric cell, they migrate to the cathode of the cell, hence, their name cations. The chief cations of the body fluids include sodium, potassium, calcium and magnesium. An important cation, present in such extremely minute amounts that we do not usually consider it, is the hydrogen ion (H^+).

Electrolytes that develop negative charges are known as "anions." An example is chloride (Cl^-). When these electrolytes are placed in a wet electric cell, they migrate to the anode, hence, the term anion. The anions of the body fluids include bicarbonate, chloride, phosphate, sulfate, organic acids and proteinate (the anion derived from plasma proteins).

There are really two types of bicarbonate:

1) Acidic bicarbonate, composed of minute amounts of bicarbonate ions "paired off" with H ions, or $H^+HCO_3^-$, and non-ionized $HHCO_3$ or H_2CO_3.

2) Base bicarbonate, which is bicarbonate "paired off" with

ions such as sodium, potassium, calcium and magnesium, or $B^+HCO_3^-$.

These two forms of bicarbonate, the former usually called "carbonic acid" and the latter "base bicarbonate," play a crucial role in body physiology, as we shall see later.

In addition to the water and electrolytes contained in the body fluids, there are certain nonionized substances such as dextrose, urea and creatinine. These are present as molecules rather than as electrically charged ions. From the standpoint of the dynamic chemical processes of the body, they are of secondary importance.

There are three properties of the body fluids that may vary in clinical fluid imbalances:

fluid volume
electrolyte composition
electrolyte concentration.

It is necessary to have suitable units of measure for fluid volume, for chemical-combining power and for osmotic pressure in order to express quantitatively variations in these properties.

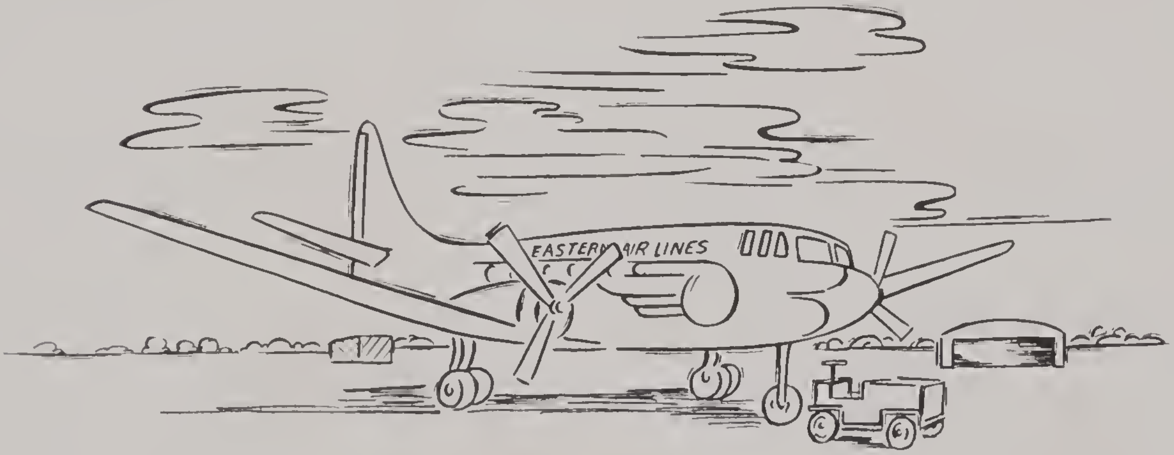
UNIT FOR VOLUME

The unit of volume is simple. The cubic centimeter has long been employed as a measure of volume. Since the centimeter is a linear measurement, it appears better, for consistency's sake, to use a volume measurement. In this handbook we will employ the milliliter which is 1/1000 of a liter. The milliliter, for all practical purposes, has the same volume as the cubic centimeter. It is abbreviated "ml."

UNIT FOR CHEMICAL-COMBINING POWER

Next, we must choose a unit of measure for chemical-combining power. The logical unit of measure for this purpose is the milliequivalent. It is abbreviated "mEq." For many years electrolytes have been measured in milligrams. The milligram, however, is a *weight* measurement and bears no relation to *chemical-combining power*, which is what we wish to measure. If one were interested in comparing the power of the engines of a locomotive with the

power of those of an airplane, one would not evaluate them on a basis of weight. Rather, one would use horsepower. A locomotive, the C. & E. I. Road Diesel, weighs 230,000 pounds but has only 1,500 horsepower. An airplane, the Eastern Airlines Martin 404, weighs only 45,000 pounds but has 4,800 horsepower.



It is obviously *horsepower,* not weight, that expresses the *power* of these two types of transportation. It is *chemical-combining power,* not *weight,* which is important when one compares electrolytes. Chemical-combining power is the same as electrical-balancing power, for chemistry and electricity are inseparably bound in the body fluids. The milliequivalent might be said to represent the "horsepower" of a given electrolyte. It can be defined as that weight of an element or ion which will combine with, or be equivalent in chemical-combining power to, 1/1000 of a gram of hydrogen. Just as our old friend the horse was the original unit of power for horsepower, so the chemical-combining power of 1/1000 of a gram of hydrogen is the unit of chemical-combining power for the milliequivalent.

An interesting example that shows the great disparity between the *weight* of an ion and its *chemical-combining power* is provided by comparing sodium and protein. A liter of plasma contains 3260 mg. of the cation sodium. This amount of sodium has a chemical-combining power of 142 mEq. On the other hand, a liter of plasma contains 65,000 mg. of the anion proteinate. This amount of proteinate has a chemical-combining power of only 16 mEq. From the standpoint of chemical-combining power, sodium is a sort of airplane, and protein a locomotive.

One milliequivalent of hydrogen has the chemical-combining power of 1/1000 of a gram of hydrogen as does 1 mEq. of sodium, potassium, chloride or phosphate. Similarly, 50 mEq. of hydrogen has the chemical-combining power of 50/1000 of a gram of hydrogen as does 50 mEq. of sodium, potassium, chloride or phosphate. Equal numbers of milliequivalents of cations "pair off" chemically with the same number of milliequivalents of anions. There is no fixed relationship between the *weight* of cations and anions that are chemically equivalent.

We like the dance analogy suggested by Statland. If we were giving a dance for teenagers, it would be ridiculous to invite 1,400 pounds of girls and 1,400 pounds of boys. We would create an unhappy situation since it is the number of individuals, not their weight, that is important from the standpoint of boys and girls pairing off for a dance.

The dance analogy has further usefulness. Let us suppose that the boys and girls were invited in equal number, and as individuals, not as dated couples. Thus, any boy could dance with any girl, and any girl with any boy. So it is with our cations and our anions. They exist largely as ions rather than as compounds such as potassium chloride or sodium phosphate. In the mad whirl of the body fluids any cation can "dance" with any anion. All that is required from the chemical standpoint is that the total cations in milliequivalents equal the total anions in milliequivalents. The compositions of the cation column and of the anion column are relatively constant in health. They may, and frequently do, vary from the norm in clinical fluid imbalances.

Why Use Milliequivalents?

There are many advantages to the use of milliequivalents in clinical fluid balance problems. One hundred milliequivalents of cations always react with exactly 100 mEq. of anions. On the other hand, there is no constant relationship between the *weight* of cations that will react with a *weight* of anions. For example, the total cations of the plasma have a milliequivalent value of 154 mEq. per liter, and they are exactly equivalent in chemical-combining power to the total plasma anions, which also have a milliequivalent value of 154 mEq. per liter. Now compare the weight of the total plasma cations with that of the total plasma anions. The former weighs 358 mg. per 100 ml. while the total plasma anions weigh 6,948 mg. per 100 ml.

The use of milliequivalents per liter as opposed to milligrams per 100 ml. is not only more logical and more realistic, but it also simplifies the calculations required for therapy. It reduces the possibility for error in such calculations. It helps in the graphing of electrolyte imbalances. Finally, it enables one to calculate the approximate level of a cation without a laboratory determination if he knows certain anion values. For example, the approximate plasma level of the cation sodium can be determined by adding the plasma bicarbonate level and the plasma chloride level plus 12, provided all the electrolyte values are expressed as milliequivalents per liter.

Conversion of Milligrams to Milliequivalents

Since many laboratory reports express electrolyte values in terms of milligrams per 100 ml., it is useful to be able to convert such weights to milliequivalents per liter. This can readily be done by use of the following formula:

$$\text{mEq./L.} = \frac{\text{mg./100 ml.} \times 10 \times \text{valence}}{\text{atomic weight}}$$

The valence and atomic weight of any element can be found in a medical dictionary or chemical text. The practitioner can quickly convert milligrams per 100 ml. to milliequivalents per liter by reference to the *conversion table* on the inside front cover and inside back cover of this handbook. He can thus avoid the necessity for mathematical conversion.

Unit for Osmotic Pressure

This brings us to the matter of "osmosis." Most of the membranes of the body are "semipermeable" membranes. Such membranes permit free passage of certain chemical molecules, such as those of water, but either partially or completely prevent the passage of other molecules, such as dextrose or electrolytes. Typical examples of semipermeable membranes are the walls of the capillaries and of the cells of the body.

Suppose that a fluid containing a relatively small number of dissolved particles is placed on one side of a semipermeable membrane, and a fluid containing a relatively large number of dissolved particles is placed on the other side. A force comes into play which equalizes the concentration of the dissolved materials in the two fluids. This force is known as "osmotic pressure."

Osmotic pressure in action is called "osmosis." Osmosis might be considered a drawing power for water, exerted by concentrated solutions on one side of a semipermeable membrane drawing water from dilute solutions on the other side. This drawing power is entirely dependent on the relative concentrations of the nondiffusible particles in the separated solutions. Obviously, when osmosis occurs, the volume of the more concentrated solution will increase as water is drawn through the semipermeable membrane from the less concentrated solution. Osmosis represents a sort of "share the water" program.

The unit of osmotic pressure is known as the "milliosmol." For many substances, it is identical with their milliequivalent values. In the case of other substances, it is different. Nevertheless, the milliequivalent can be used as a rough, though adequate, measurement of osmotic pressure as well as of chemical-combining power. Thereby, needless calculations and confusion are avoided.

SUMMARY

1) All body fluids consist of water and important dissolved substances.

2) Of the dissolved substances, we are especially interested in those active, dynamic chemicals called electrolytes, which develop electrical charges when placed in water.

3) Electrolytes that develop positive charges are called *cations;* those that develop negative charges are called *anions.*

4) Certain nonionized substances such as dextrose, urea and creatinine are present in the body fluids as molecules.

5) In order to study changes that occur in the body fluids, one must have units of measure for fluid volume, for chemical-combining power of individual electrolytes, and for osmotic pressure.

6) The unit of measure for *volume* is the *milliliter,* defined as 1/1000 of a liter. It is abbreviated "ml."

7) The unit of measure for *chemical-combining power* is the *milliequivalent,* defined as the chemical-combining power of 1/1000 of a gram of hydrogen. It is abbreviated "mEq."

8) The *milliequivalent* may also be used as the unit of measure for *osmotic pressure* for clinical purposes.

5

Composition of Body Fluids

Nothing but a cloud of elements organic, . . . Such as man is made of.

Oliver Wendell Holmes

Each of the three major types of body fluid—the plasma, the interstitial fluid and the cellular fluid—has its own normal composition of cations and anions. The cations of plasma and of interstitial fluid include sodium, potassium, calcium and magnesium. *Sodium is the chief cation of the plasma and of the interstitial fluid.* The anions of plasma and of interstitial fluid include bicarbonate, chloride, phosphate, sulfate, organic acids and proteinate. *Chloride is the chief anion of the plasma and of the interstitial fluid.* The significant difference between the plasma and the interstitial fluid is the much greater quantity of proteinate contained in the plasma (see plates I and II).

The cations of the cellular fluid include sodium, potassium, calcium and magnesium. *Potassium is the important cation of the cellular fluid.* The anions of the cellular fluid include bicarbonate, chloride, phosphate, sulfate and proteinate. *Phosphate is the important anion* (see plate III).

The total cations of any body fluid always equal the total anions of that same body fluid, since electrical balance must be maintained. It is important to recognize that electrolytes exist in the body fluids not as *compounds* such as potassium chloride or sodium phosphate, but rather as *independent cations and anions.* They exist in electrical balance but not in union with one another. They can be thought of as dashing madly about in the great chemical reaction vat of the body fluids, maintaining the electrical balance of power but determinedly avoiding entangling alliances.

SUMMARY

1) Each major type of body fluid in health has a characteristic composition of cations and anions.

2) The cations of a given fluid always equal the anions when both are expressed as milliequivalents.

3) Electrolytes do not occur in the body fluids as compounds but rather as independent cations and anions which are in electrical balance.

6

Gains and Losses of Body Fluids

> *(Diseases) . . . attenuate our bodies, dry them, wither them, shrivel them up like old apples, make them so many anatomies.*
>
> ROBERT BURTON

ABNORMAL differences between the gains and losses of body fluids lie at the roots of most clinical fluid imbalances. Fluids, composed of water and electrolytes in various proportions, are gained in the following ways:

- ingested water
- ingested food
- tube feedings
- parenteral feedings
- rectal feedings

Water alone is gained through:

- oxidation of foodstuffs
- oxidation of body tissues

The list of routes through which fluids are lost is far longer. *Water and electrolytes* may be lost from the:

- Eyes in
  - lacrimation
- Skin in
  - perspiration
  - burn exudate
  - wound exudate
  - decubitus ulcer exudate
- Mammary glands in
  - breast milk
- Vascular system in
  - hemorrhage
- Lymphatic system in
  - lymphatic duct fistulas
- Respiratory tract in
  - bronchorrhea
  - pulmonary edema
- Gastrointestinal tract in
  - salivation
  - vomiting
  - gastric suction
  - intestinal fistulas
  - enterostomies
  - cecostomies
  - colostomies
  - normal stools
  - diarrheal stools

Genitourinary tract in
  urine
Central nervous system in
  cerebrospinal fluid from ventricular or spinal drainage
Water and electrolytes also may
  be lost because of
  draining abscesses
  thoracentesis
  paracentesis
  internal losses as
    pooling
  fixation of certain electrolytes by secretions
  edema formation in injured or burned areas
  massive urticaria.
Water alone is lost from the
  skin as
    insensible perspiration
  respiratory tract as
    water vapor during expiration (see plate IV).

The state of fluid balance of the healthy adult can readily be determined by comparing the *volume of fluid consumed, as fluid,* with the *volume of urine.* Normally these are approximately equal. This simple rule holds because the other chief sources of gain of fluid in health,

1) Water derived from solid food, and

2) Water obtained from chemical oxidation in the body approximately equal the normal losses:

1) Water lost as vapor through the lungs and skin, and

2) Water lost in the stools.

This rule does not apply in sickness to infants, children or adults. In sickness, when ingestion of fluid as fluid and as solid food ceases or is diminished, the gains are overbalanced by the losses. Then the patient may readily lose half a liter a day more than he gains. Daily losses are cumulative and a serious deficit in fluid volume can develop fairly quickly, even when abnormal losses in body fluids such as those of diarrhea or vomiting are not occurring. When such abnormal losses do occur, the patient can be placed at death's door in a matter of days or even hours.

Losses of body fluids through such routes as fistulas, intestinal suction, vomiting, ileal or cecal stomata, diarrhea or sweating involve not only loss of water but also of varying amounts of electrolytes, according to the composition of the fluid lost. *The body secretions and excretions vary widely, both in their total concentration of electrolytes and in their electrolyte composition.* Comparison of the electrolyte composition of important body secretions and excretions with that of plasma is shown in simple bar charts (see plates V, VI and VII). The last bar chart on plate VII shows the average daily loss of sodium, potassium and chloride in diarrheal stools of children. Losses of bicarbonate are not shown in this bar chart because quantitative data on such losses are not available. Obviously, both the volume and the electrolyte composition of a secretion or excretion lost are important.

SUMMARY

1) Discrepancies between gains and losses of body fluids are responsible for most clinical fluid imbalances.

2) The number of routes through which water and electrolytes are lost is far greater than the number through which they are gained.

3) The state of fluid balance of the healthy person can be assessed by comparing the volume of fluid consumed as fluid with the volume of urine. This rule does not apply to ill infants, children or adults, since in sickness, when ingestion of food and liquids ceases or diminishes, the losses outweigh the gains.

4) Fluids lost from the body vary widely in their electrolyte composition; hence, both the volume and the source of fluid losses are important in the evaluation of the clinical condition of the patient.

7

Body Automation

It was through the evolution of this filtration-reabsorption system that the kidney came to be almost entirely responsible for the composition of the internal environment of the body, manufacturing it, as we have said, in reverse, by saving some things from the glomerular filtrate and rejecting others. . . .

HOMER W. SMITH

A CENTURY and a half ago, a strange German religious sect, the Rappites, founded the community of Harmonie on the banks of the Wabash. They built well and their sturdy, ivy-covered brick buildings still stand. Their leader was George Rapp, and therein lies our tale, for the name R A P P spells out the first letters of the end organs which provide automatic normalization or homeostasis of the fluid volume, electrolyte concentration and electrolyte composition of the body fluids. These homeostatic controls include the *R*enal, *A*drenal, *P*ituitary and *P*arathyroid mechanisms.

They are key units of a fantastically complicated regulatory system which puts man-made automation to shame.

Mention should be made of the lungs which may also be regarded as organs of body homeostasis, since they regulate the carbonic acid level of the extracellular fluid through exhalation or retention of carbon dioxide. Their role, both in the correction and causation of body fluid disturbances, is covered in detail in Chapter 11.

The *renal mechanism*, consisting of the kidneys, acts both autonomously and in response to blood-borne influences to regulate the water and electrolyte content of body fluids. It does this through adjustments in the volume, concentration and composition of the urine. Amazingly efficient in most respects, the renal mechanism *appears* to have a strange disregard for conservation of potassium, which is excreted even when a severe deficit of this cation exists.

Since the kidneys are completely dependent for the exercise of their regulatory functions upon the heart and blood vessels, the renal mechanism is sometimes appropriately referred to as the *renocardiovascular mechanism*. We shall employ the shorter term, *renal mechanism*, for convenience.

Impairment of the renal mechanism occurs in the following:

- renal ischemia due to
    - critical fall in systolic blood pressure
    - heart failure
    - volume deficit of extracellular or body fluid
    - thrombosis or embolism of renal artery or vein
- acute glomerulonephritis
- advanced chronic glomerulonephritis
- advanced chronic pyelonephritis
- severe arteriolar nephrosclerosis
- lower nephron nephrosis
- childhood lipoid nephrosis
- smaller premature infants during the first few days of life
- toxemia of pregnancy
- postoperative phase of surgery, secondary to
    - volume deficit of extracellular or body fluid
        - due to fasting,

antidiuretic action of narcotics such as morphine
retention of sodium due to "alarm" reaction.

The *adrenal mechanism* is intimately associated with retention or excretion of sodium, potassium and water. It apparently exerts these effects through the action of the adrenocortical hormones on the kidneys. In adrenal insufficiency there is characteristically a tendency toward:

elevated plasma potassium
depressed plasma sodium
inability to excrete a water load.

In adrenal hyperactivity there is a tendency toward:

excessive sodium retention with resultant edema
excessive urinary loss of potassium with depression of plasma potassium levels.

Impairment of the adrenal mechanism occurs in:

1) Insufficiency of the adrenal cortex, due to:
    - Addison's disease
    - congenital adrenal hyperplasia
    - diminished production of endogenous ACTH
    - sudden withdrawal of exogenous ACTH or of synthetic adrenocortical hormones such as cortisone,
2) Hyperactivity of the adrenal cortex, due to:
    - overproduction of endogenous ACTH
    - overdosage of exogenous ACTH
    - tumor of the adrenal cortex
    - primary aldosteronism
    - overdosage with synthetic adrenocortical hormones such as cortisone or desoxycorticosterone acetate (DOCA).

The *pituitary mechanism* comprises both the anterior and the posterior pituitary glands. The *anterior pituitary gland* secretes a number of hormones, some of which exert a direct effect on the metabolism of water and electrolytes. Others exert an indirect effect by stimulating other endocrine glands whose hormones

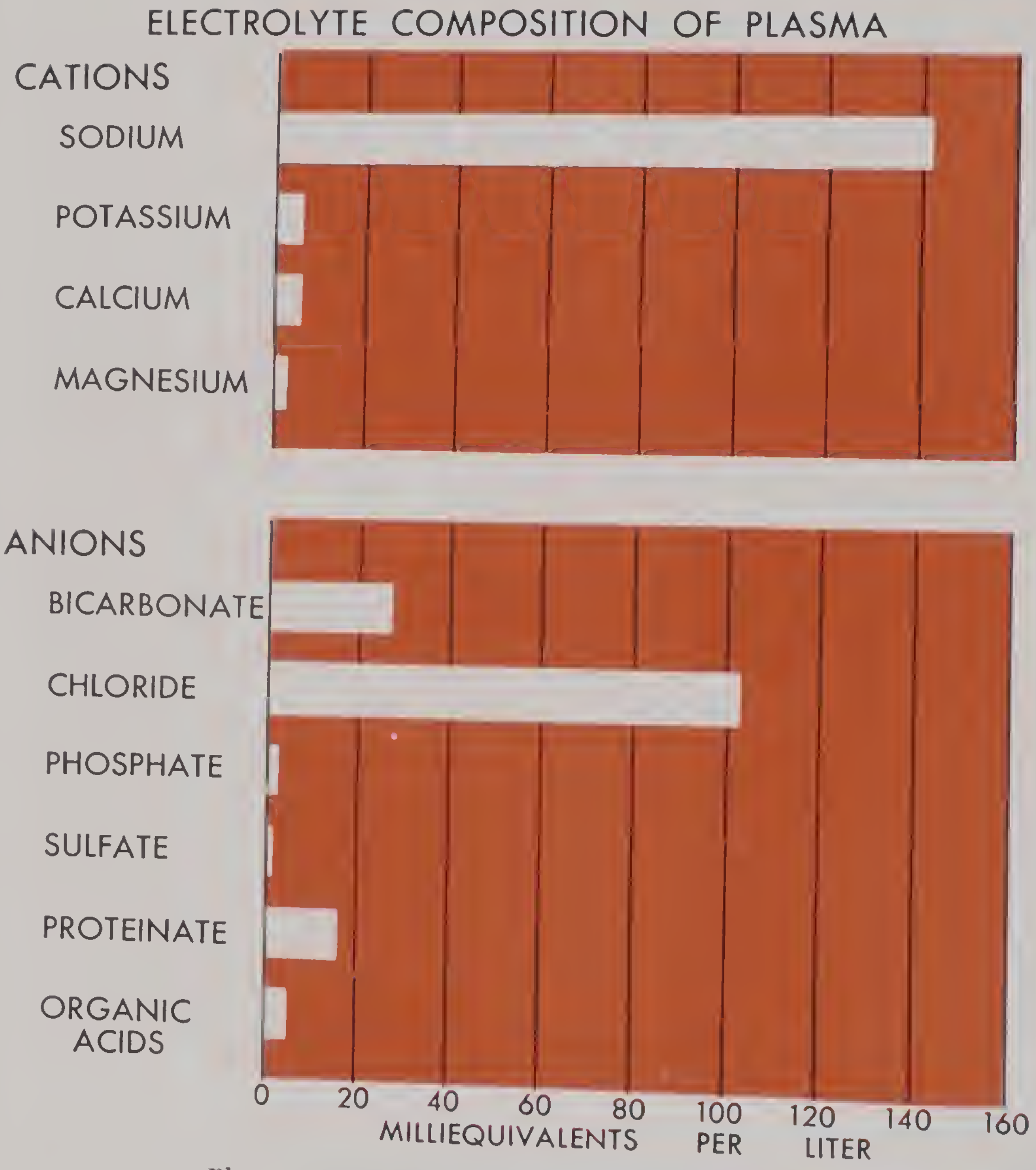


Plate I. Electrolyte Composition of Plasma.

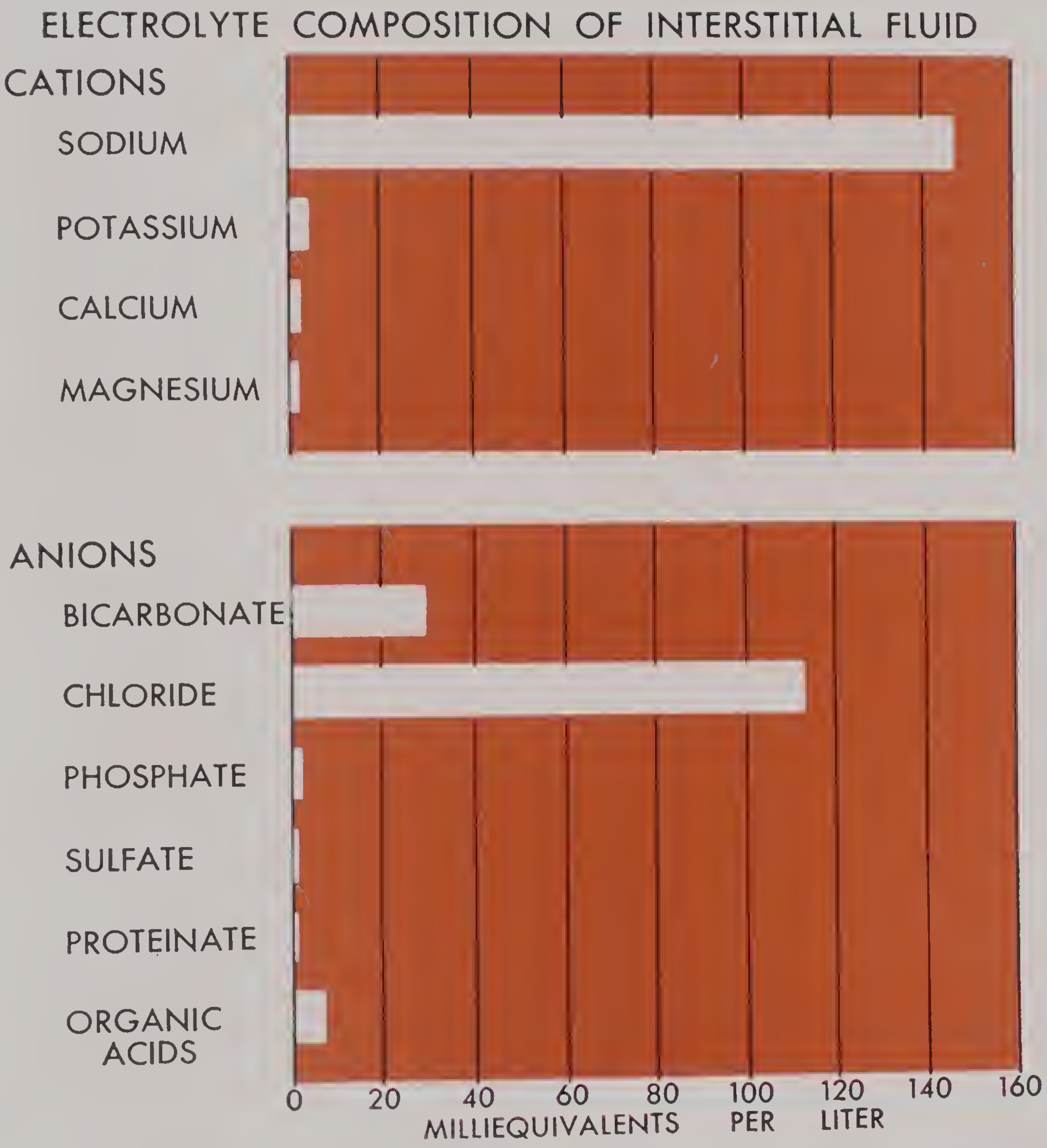


Plate II. Electrolyte Composition of Interstitial Fluid.

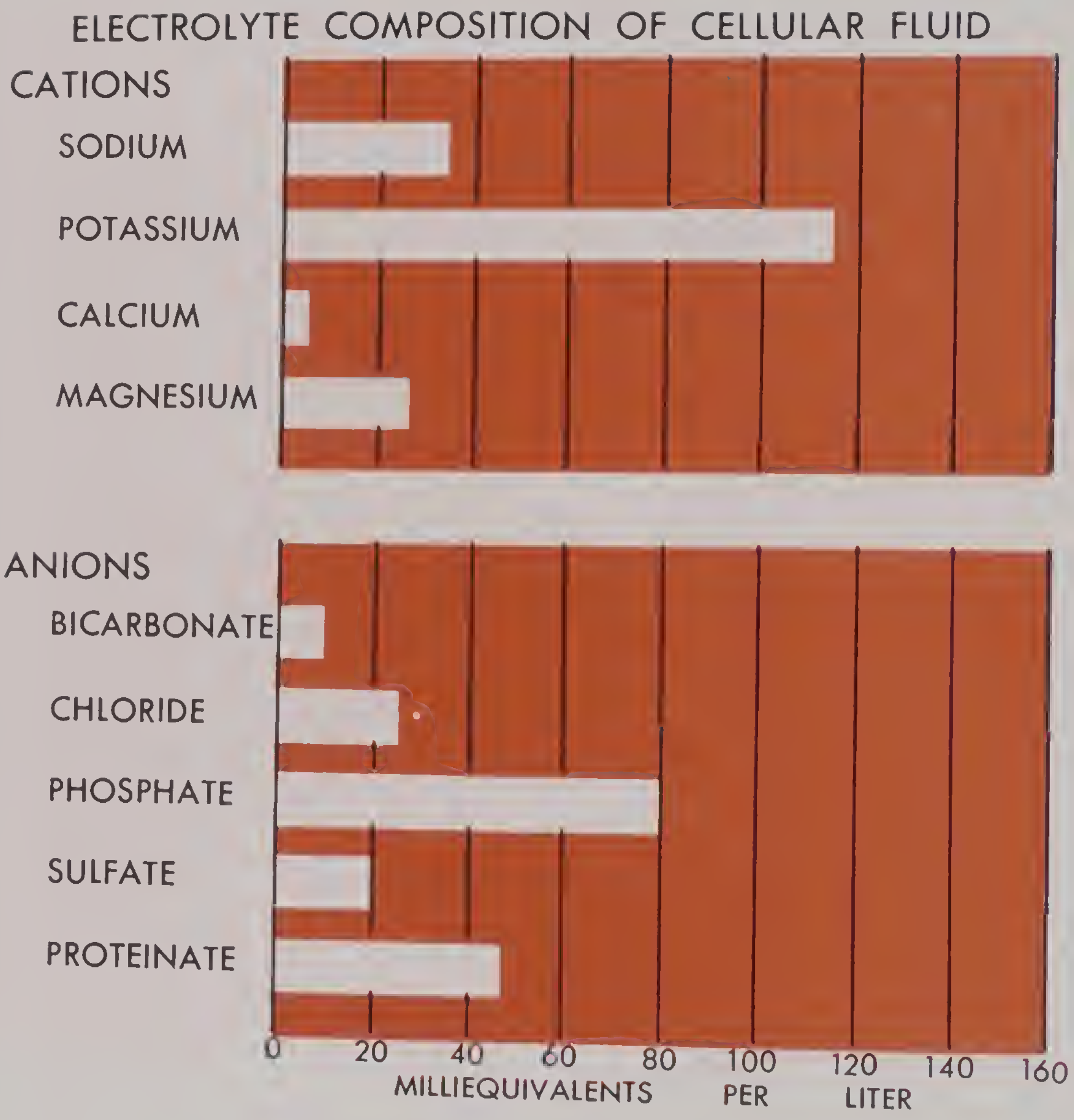


Plate III. Electrolyte Composition of Cellular Fluid.

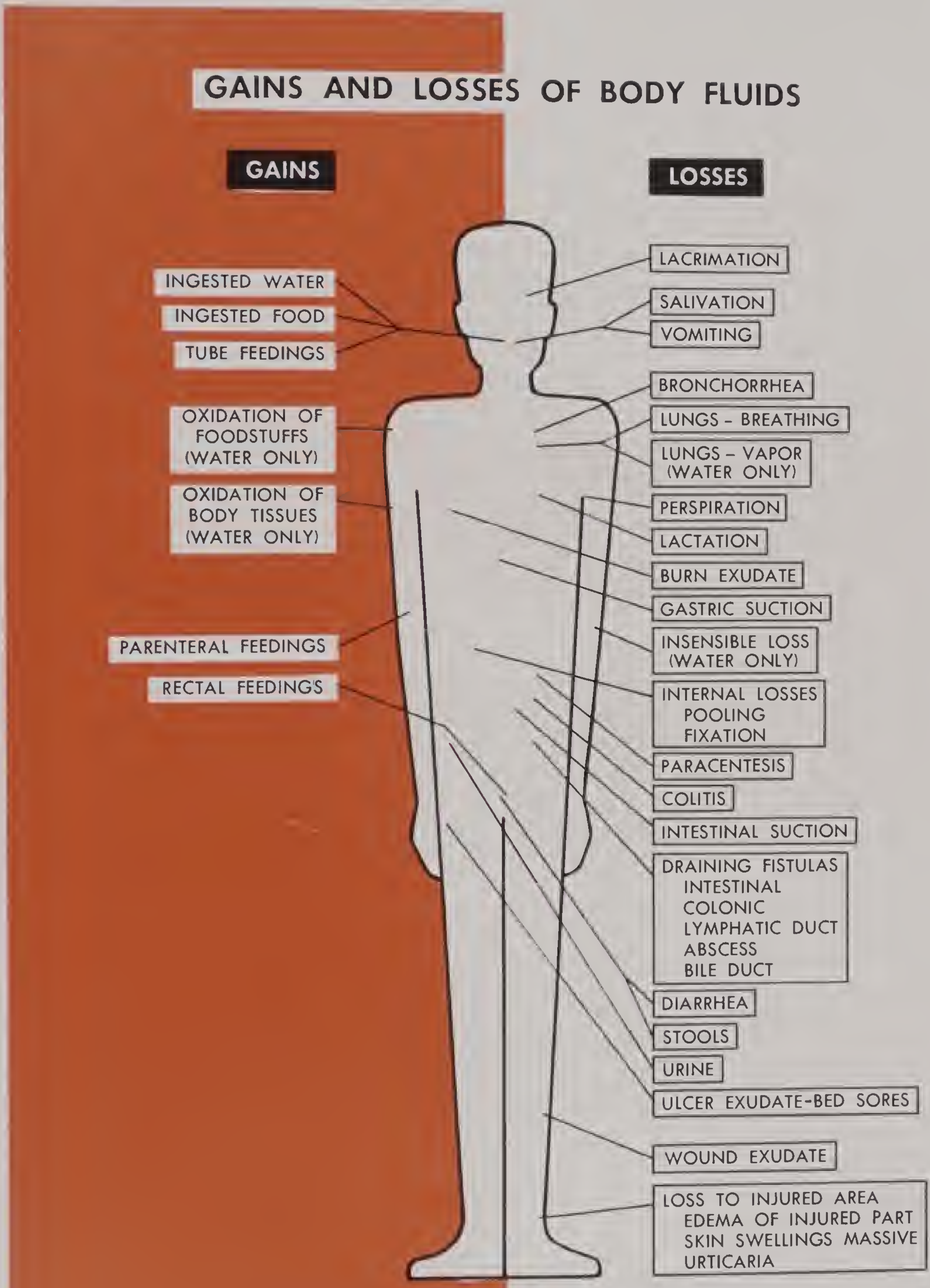

GAINS AND LOSSES OF BODY FLUIDS
GAINS
LOSSES
INGESTED WATER
INGESTED FOOD
TUBE FEEDINGS
OXIDATION OF FOODSTUFFS (WATER ONLY)
OXIDATION OF BODY TISSUES (WATER ONLY)
PARENTERAL FEEDINGS
RECTAL FEEDINGS
LACRIMATION
SALIVATION
VOMITING
BRONCHORRHEA
LUNGS – BREATHING
LUNGS – VAPOR (WATER ONLY)
PERSPIRATION
LACTATION
BURN EXUDATE
GASTRIC SUCTION
INSENSIBLE LOSS (WATER ONLY)
INTERNAL LOSSES POOLING FIXATION
PARACENTESIS
COLITIS
INTESTINAL SUCTION
DRAINING FISTULAS INTESTINAL COLONIC LYMPHATIC DUCT ABSCESS BILE DUCT
DIARRHEA
STOOLS
URINE
ULCER EXUDATE-BED SORES
WOUND EXUDATE
LOSS TO INJURED AREA EDEMA OF INJURED PART SKIN SWELLINGS MASSIVE URTICARIA

Plate IV. Gains and Losses of Body Fluids.

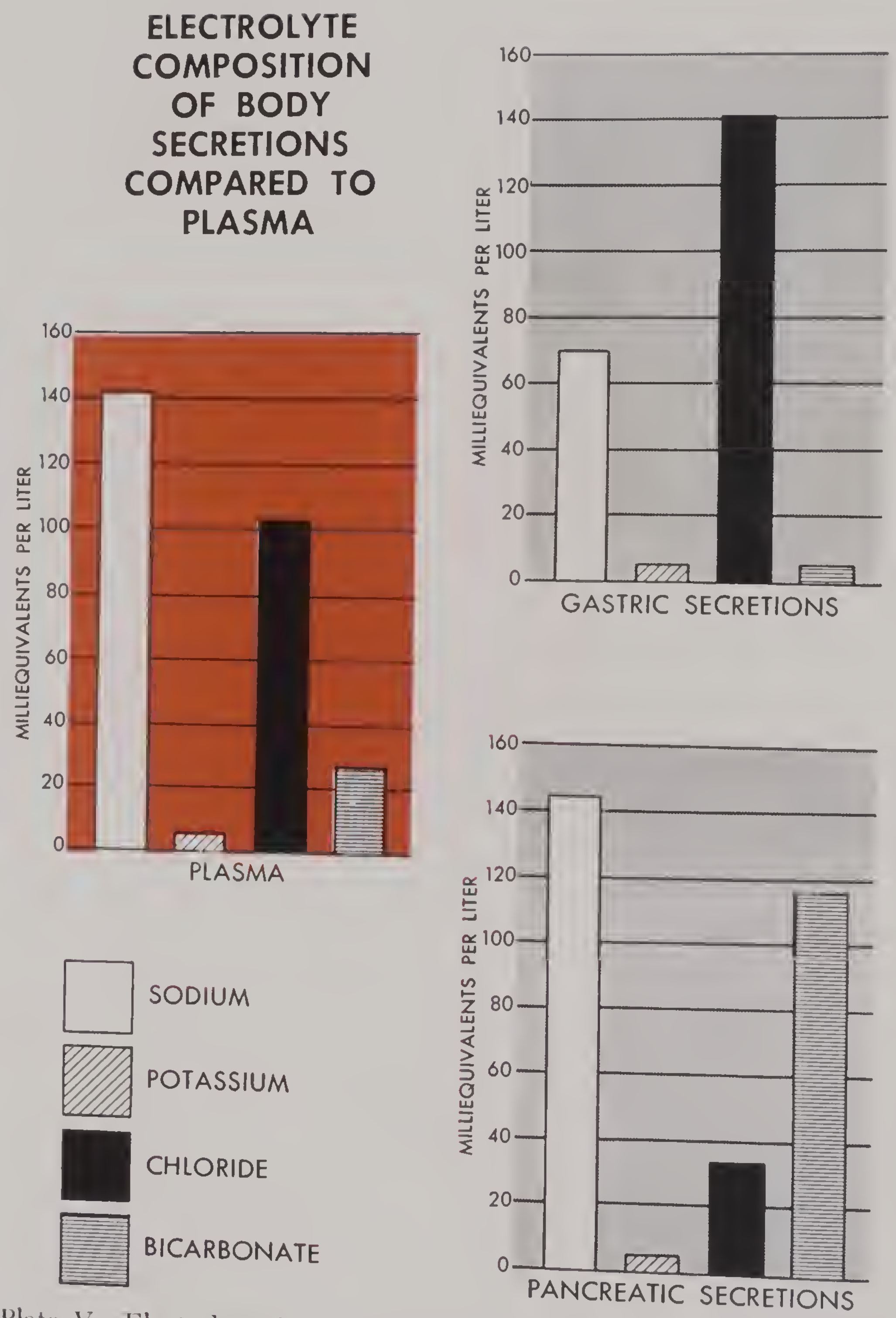


Plate V. Electrolyte Composition of Body Secretions Compared to Plasma (1).

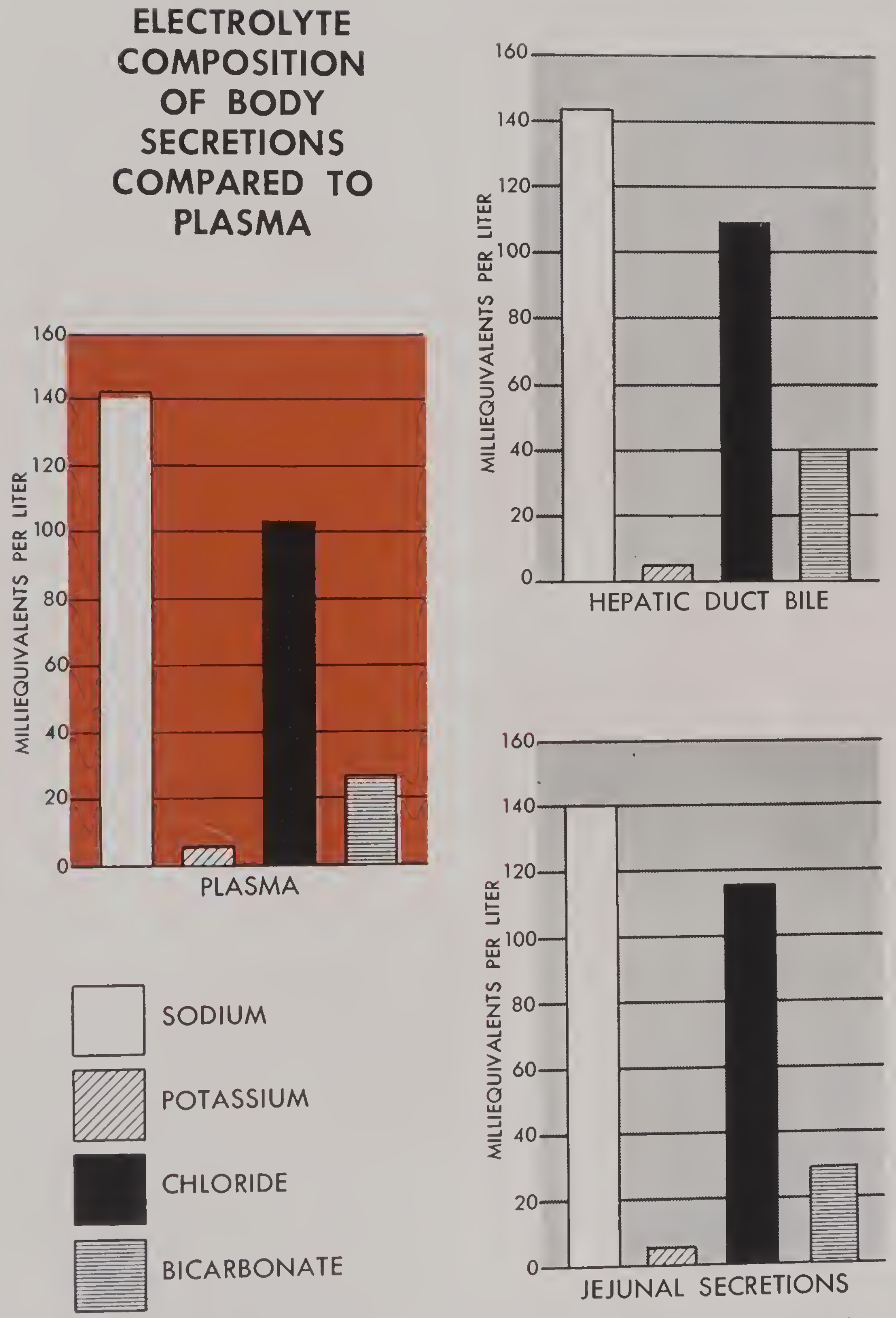


Plate VI. Electrolyte Composition of Body Secretions Compared to Plasma (2).

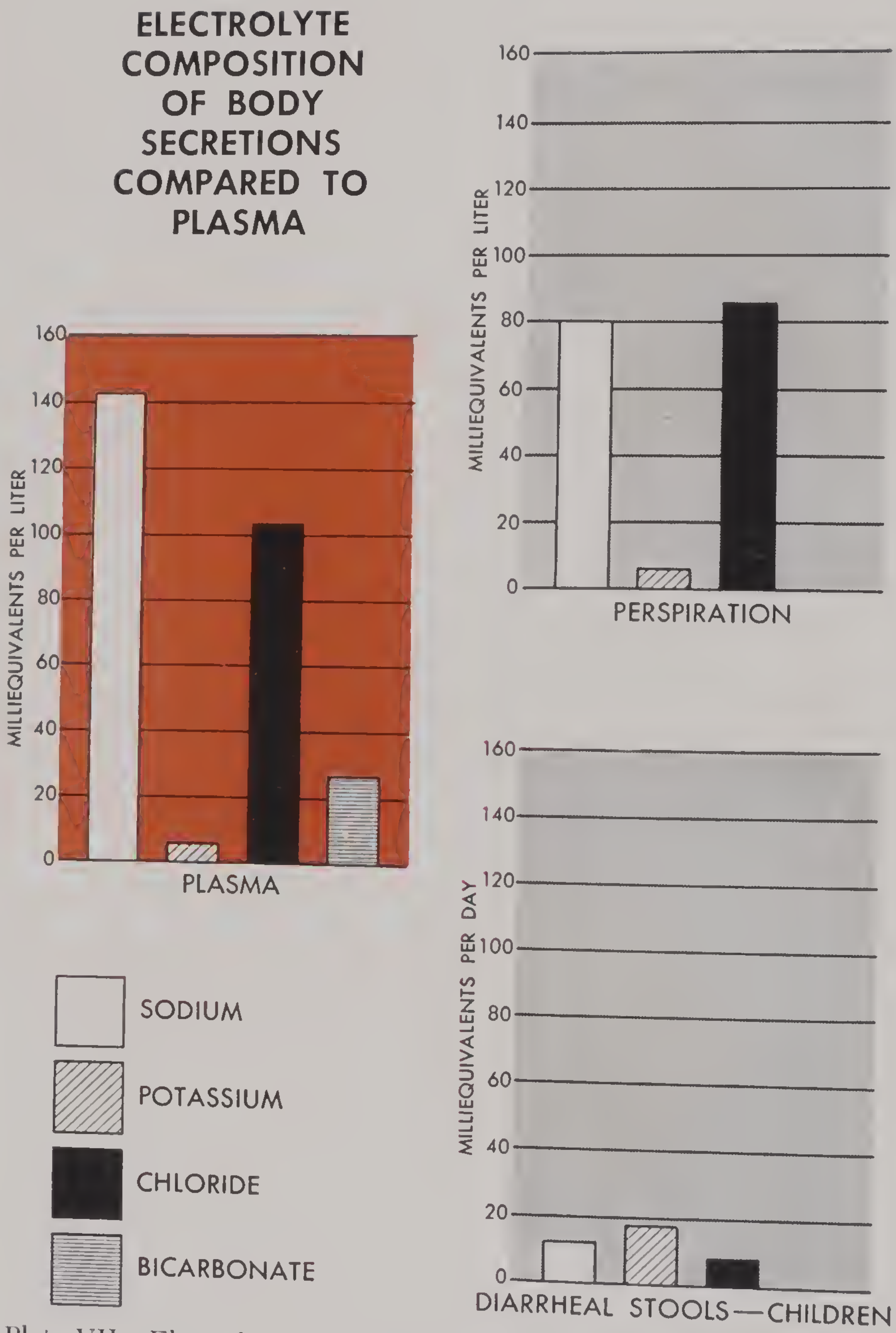


Plate VII. Electrolyte Composition of Body Secretions Compared to Plasma (3).

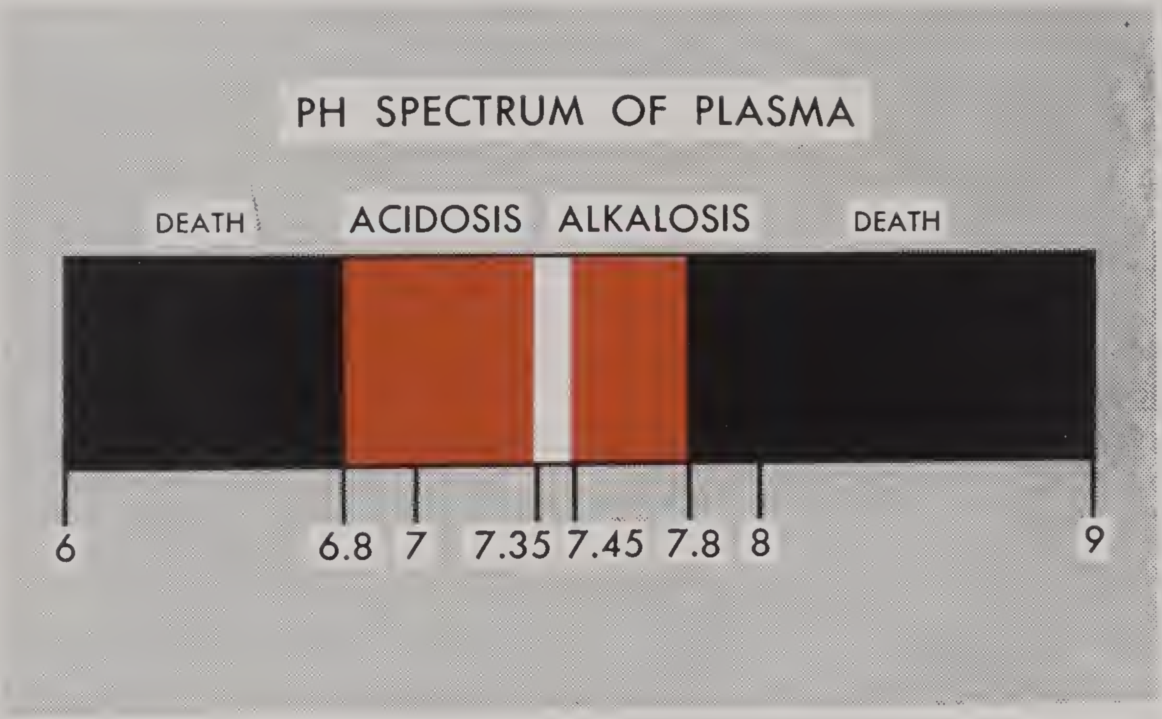


Plate VIII. pH Spectrum of Plasma.

directly influence metabolism. Pituitary growth hormone exerts a direct effect. Other hormones of the anterior pituitary gland, including thyroid stimulating hormone (TSH), adrenocortical stimulating hormone (ACTH), and the gonadotropic hormones, are indirect in their action.

Excessive or deficient production of a single anterior pituitary hormone is rare. Examples of overproduction of a single hormone include overproduction of growth hormone, which results in acromegaly, and overproduction of ACTH, which results in Cushing's syndrome. Depressed function of the anterior pituitary gland usually involves all of its hormones. It is seen in such clinical syndromes as congenital or acquired panhypopituitarism and Simmond's disease (pituitary cachexia). Underproduction of a single hormone occurs following sudden withdrawal of such therapeutically supplied hormones as ACTH.

The *posterior pituitary gland* releases a water-conserving hormone when stimulated by action of the "osmostat," an auxiliary control located in the plexus of the internal carotid artery. The osmostat is sensitive to changes in the electrolyte concentration of the extracellular fluid. This water-conserving hormone is usually referred to as the "antidiuretic hormone" or "ADH." As the name implies, it *inhibits diuresis.* It seems more forthright to think of it as *conserving water.* Production of the water-conserving hormones ceases in the disease diabetes insipidus, which affects the posterior pituitary gland. The result is polyuria. An inability to concentrate the urine under conditions of fluid deficit is diagnostic of diabetes insipidus. Production of the water-conserving hormone is also suppressed in a number of physiologic conditions.

Impairment of the anterior pituitary mechanism occurs in:

1) Underproduction of a pituitary hormone, as in:
    panhypopituitarism, either congenital
        or acquired,
    Simmond's disease, and in
    sudden withdrawal of therapeutically supplied
        hormones such as ACTH,
2) Overproduction of a pituitary hormone, as in:
    Cushing's syndrome, and acromegaly.

Impairment of the posterior pituitary mechanism occurs in:

diabetes insipidus
emotional stress
excessive drinking of water
ingestion of alcohol
following exposure to cold
morphine administration

The parathyroid mechanism maintains the level of ionized calcium in the blood. It fails to do so when excessive amounts of parathyroid are accidentally removed in thyroidectomy, in primary hypoparathyroidism, or in pseudohypoparathyroidism. Hypoparathyroidism is characterized by a decrease in ionized calcium in the plasma, a decrease in the total plasma calcium level, and tetany. The opposite condition, hyperparathyroidism, may be caused by neoplasms of the parathyroid glands or by primary hyperparathyroidism. It is manifested clinically by polyuria, deposit of calcium in the kidneys, and damage to the renal tubules.

Impairment of the parathyroid mechanism occurs in:

1) Underproduction of parathyroid hormone, as in surgical removal of parathyroid gland, and in primary hypoparathyroidism.
2) Failure of end organs to react to parathyroid hormone, as in pseudohypoparathyroidism.
3) Overproduction of parathyroid hormone, as in primary hyperparathyroidism, and in tumors of parathyroid gland.

So long as the renal, adrenal, pituitary and parathyroid homeostatic mechanisms are functioning, the body tolerates and adapts to variations in intake and output of water and electrolytes amazingly well. We shall see how advantage can be taken of this inherent body automation in the chapters on therapy, which describe parenteral solutions that permit the body, in its wisdom, to selectively retain or reject water and electrolytes according to its needs. We shall also see that when the body homeostatic mechanisms are impaired, as in the pathologic states above listed, one can no longer rely on them. Then the physician must "tailor" the parenteral fluid regime to meet what he estimates as the body's needs. The patient must exchange the wisdom of the body for the wisdom of the physician, often a poor exchange indeed.

SUMMARY

1) The homestatic controls of the body provide automatic normalization of the fluid volume, electrolyte concentration and electrolyte composition of the body fluids. These controls include the renal, adrenal, anterior and posterior pituitary and parathyroid mechanisms.

2) The renal mechanism regulates the water and electrolyte content of the body fluids through adjustments in the volume, concentration and composition of the urine.

3) The adrenal mechanism is closely associated with retention or excretion of sodium, potassium and water.

4) The anterior pituitary gland secretes several hormones. Some of these exert their effects directly on the metabolism of water and electrolytes; others exert indirect effects by stimulation of other endocrine glands.

5) The posterior pituitary gland releases a water-conserving hormone, the antidiuretic hormone.

6) The parathyroid mechanism maintains the level of ionized calcium in the blood.

7) As long as the renal, adrenal, pituitary and parathyroid homeostatic controls function, the body tolerates and adapts to variations in intake and output of water and electrolytes over wide ranges.

8

The Mystery of the Cells

> *The sea never changes and its works, for all the talk of men, are wrapped in mystery.*
>
> JOSEPH CONRAD

ALTHOUGH the cellular fluid and the extracellular fluid are in osmotic equilibrim, they are, as we have seen, strikingly different in their electrolyte composition. Sodium is overwhelmingly the predominant cation of the extracellular fluid, potassium of the fluid within the cells. Chloride is the chief anion of the extracellular fluid, phosphate of the cellular fluid. There are other important differences. How these differences can exist in the light of what is known concerning the physical properties of the cell membranes is what Gamble has termed one of the "inscrutable phenomena." Yet this is but one of many puzzles that concern the cells.

Exchanges of water and dissolved materials between the cells and the fluid in which they are bathed are continuous and of immense magnitude. Since these exchanges can adequately be explained on the basis of no known physical or chemical processes, it is intriguing to speculate on the nature of the ghostly pumps that begin their work months before we are born and continue without respite until death supervenes.

Our knowledge of the composition of the many different types of body cells is sketchy at best. It is because of our lack of precise knowledge that we group all the body cells in one category when we describe the electrolyte composition of the cellular fluid. Truly, the works of the cells, like those of the sea, "are wrapped in mystery."

Yet we do know much of practical import. When the concentration of electrolytes in the extracellular fluid is elevated, water leaves the cells to restore osmotic equilibrium. Thus, injection or ingestion of a concentrated solution of sodium chloride causes the extracellular fluid to expand and the cellular fluid to shrink.

If, on the other hand, electrolytes are lost from the body, as happens when an individual sweats heavily and drinks only water, the electrolyte concentration of the extracellular fluid decreases. Water from the extracellular fluid then enters the cells until the electrolyte concentration is the same inside the cells as it is in the fluid surrounding the cells. Again, osmotic equilibrim is achieved. Under certain conditions the extracellular fluid volume may be so decreased that circulatory failure develops, while the cells are filled to the bursting point with water.

Since the cellular fluid comprises nearly two-thirds of the total body fluid, one would suppose that the cells would serve as an available reserve supply of water and electrolytes to compensate for acute losses of extracellular fluid. Such is not necessarily the case, since the cellular fluid appears to be functionally unavailable to the extracellular fluid in such desperate emergencies as result from massive extravasation of extracellular fluid into badly injured regions of the body.

In acute losses of extracellular fluid from the body, the cellular fluid is not appreciably affected. As losses continue, fluid is lost from the cells into the extracellular fluid. In any fluid deficit that has developed over a period of days, the loss involves not only extracellular fluid, but cellular fluid as well. One practical implication of this fact is that solutions designed for repair of body fluid deficits should contain cellular as well as extracellular electrolytes.

Just as deficits of cellular water and electrolytes develop slowly, so it takes time to repair such deficits. Correction of a severe potassium deficit, for example, may require from five to seven days. Such deficits are corrected at least as well by mouth as by parenteral administration of fluids, if the patient can take oral fluids.

David B. Coursin has compared the extracellular fluid to the visible portion of an iceberg and the cellular fluid to the greater mass of ice that lurks, invisible, beneath the water's surface. The analogy is especially appropriate since it places proper emphasis on the immense importance of the cellular fluid, at the same time indicating that knowledge of its clinical status must be derived largely from observations of the less voluminous and frequently misleading but *accessible* extracellular fluid.

Although the status of cellular affairs must never be ignored in clinical disturbances of the body fluids, it acquires special significance when an electrolyte that is primarily cellular is under consideration. For example, of the adult's 3000 mEq. of potassium, only 50 to 70 mEq. is normally contained in the extracellular fluid. Moreover, the level of potassium in the extracellular fluid is not always a reliable index of the cellular supply. Thus, the plasma potassium level may be elevated in the face of a cellular deficit; it may be depressed when a cellular excess exists. It may be normal in either instance or it may vary directly with the cellular stores.

As Moore and others have pointed out in a fascinating article, "Body Sodium and Potassium: V. The Relationship of Alkalosis, Potassium Deficiency and Surgical Stress to Acute Hypokalemia in Man," a reading *must* for the practitioner interested in understanding extracellular-cellular relationships, "In general, loss of an ion from the body (deficiency) is not synonymous with low serum concentration (hypo-emia). The two often travel together but they do not move at the same rate; they may move in opposite directions; one is not infrequently seen without the other."

Only when the plasma potassium reading is considered in the light of other data, such as those obtained from the clinical history, the physical findings, other laboratory tests and the electrocardiogram, is the plasma potassium level of real value in assessing the cellular status of the electrolyte.

Potassium is certainly the star actor in the cellular drama. It is present within the cells, both in the ionized form and in a bound form combined with protein. In the breakdown of body tissues potassium and protein are excreted in a fixed ratio. When protein is administered, potassium must be present in the feeding in adequate amounts if new tissue synthesis is to occur.

Potassium is involved in energy metabolism associated with the utilization of oxygen and dextrose within the cellular fluid. Potassium will not move into the cell if anoxemia is present, or if sufficient dextrose and insulin are not present.

SUMMARY

1) The intimate metabolism of the body cells is wrapped in mystery, yet we do know much of practical import.

2) When the concentration of electrolytes in the extracellular fluid is elevated, water leaves the cells to restore osmotic equilibrium.

3) When the electrolyte content of the extracellular fluid decreases, water from the extracellular fluid enters the cells until osmotic equilibrium is achieved.

4) The cellular fluid may be functionally unavailable to the extracellular fluid in certain acute emergencies, such as massive extravasation of extracellular fluid into badly injured parts of the body.

5) Potassium is the most important cellular electrolyte. Its level in the plasma does not always reflect the amount of potassium in the cells.

Part II Clinical Diagnosis

9

The Tower of Babel

> *. . . the name thereof was called Babel, because there the language of the whole earth was confounded . . .*
>
> GENESIS

ONE OF the most confusing aspects of the subject of clinical fluid balance has been the variations in terms used by different writers and investigators. Some authors have applied one term, others a widely divergent one to identical entities. In other instances, a term which has one meaning for one investigator has exactly the opposite meaning for another. To the clinician going from lecture to lecture, from article to article, from book to book, this inconsistency of terminology all too often resembles a jargon, a sort of twentieth century "Tower of Babel."

What is needed as a first step to clearing the air is a classification which is at once:

systematic
clear
logical
all inclusive
clinical

and, most important of all, *not susceptible to misinterpretation.* The ideal classification should be eminently practical. It should serve as a useful basis for diagnosis and treatment.

A classification meeting most of these criteria was introduced by Moyer. It has been modified somewhat and extended by the authors in an effort to make it approach as closely as possible the ideal. It will be employed throughout this handbook.

Basically, this classification divides body fluid imbalances into three basic types:

1) Those involving changes in certain *properties of extracellular fluid.*
2) Those involving changes in *location of extracellular fluid.*
3) Those involving changes in the *body's nutritional status.*

The imbalances which make up these three types will be described first in a general manner, then as specific "clinical pictures."

SUMMARY

1) Much would be gained by the universal employment of a comprehensive, practical and clear classification of the clinical fluid imbalances.

2) Moyer has introduced such a classification.

3) Moyer's classification, as modified and extended by the authors, divides body fluid imbalances into three basic types, those involving changes in:

a) Properties of extracellular fluid.
b) Location of extracellular fluid.
c) Nutritional status of the body.

10

Specific Fluid Imbalances: Three Basic Types

> *System, or as I shall term it, the virtue of method, is the harness without which only the horses of genius travel.*
>
> Sir William Osler

The first basic type of fluid imbalance is characterized by changes in the *properties* of the extracellular fluid. This type includes *volume* changes, either deficit or excess; electrolyte *concentration* changes, either deficit or excess; changes in the electrolyte *composition* of the extracellular fluid, including potassium deficit or excess; calcium deficit or excess; base bicarbonate deficit or excess, and carbonic acid deficit or excess.

The second basic type of imbalances includes those due to the *position* of extracellular fluid. There are two such specific fluid imbalances. The first is characterized by a shift of water and electrolytes from plasma to interstitial fluid. The second is caused by a shift of water and electrolytes from interstitial fluid to plasma.

The third basic type of specific fluid imbalances includes those characterized by changes in the *nutritional status* of the body as a whole.

GENERAL DESCRIPTION OF THREE BASIC TYPES

Changes in the properties of extracellular fluid are largely reflected in the plasma. Since the plasma is the portion of the extracellular fluid which is readily accessible to us, let us consider changes in volume, concentration and composition of extracellular fluid as reflected in the plasma. We will use a simple picture analogy in which we imagine that all of the plasma of the body is contained in a beaker (see Figure 5).

Notice the upper row of beakers, titled "volume." The left-

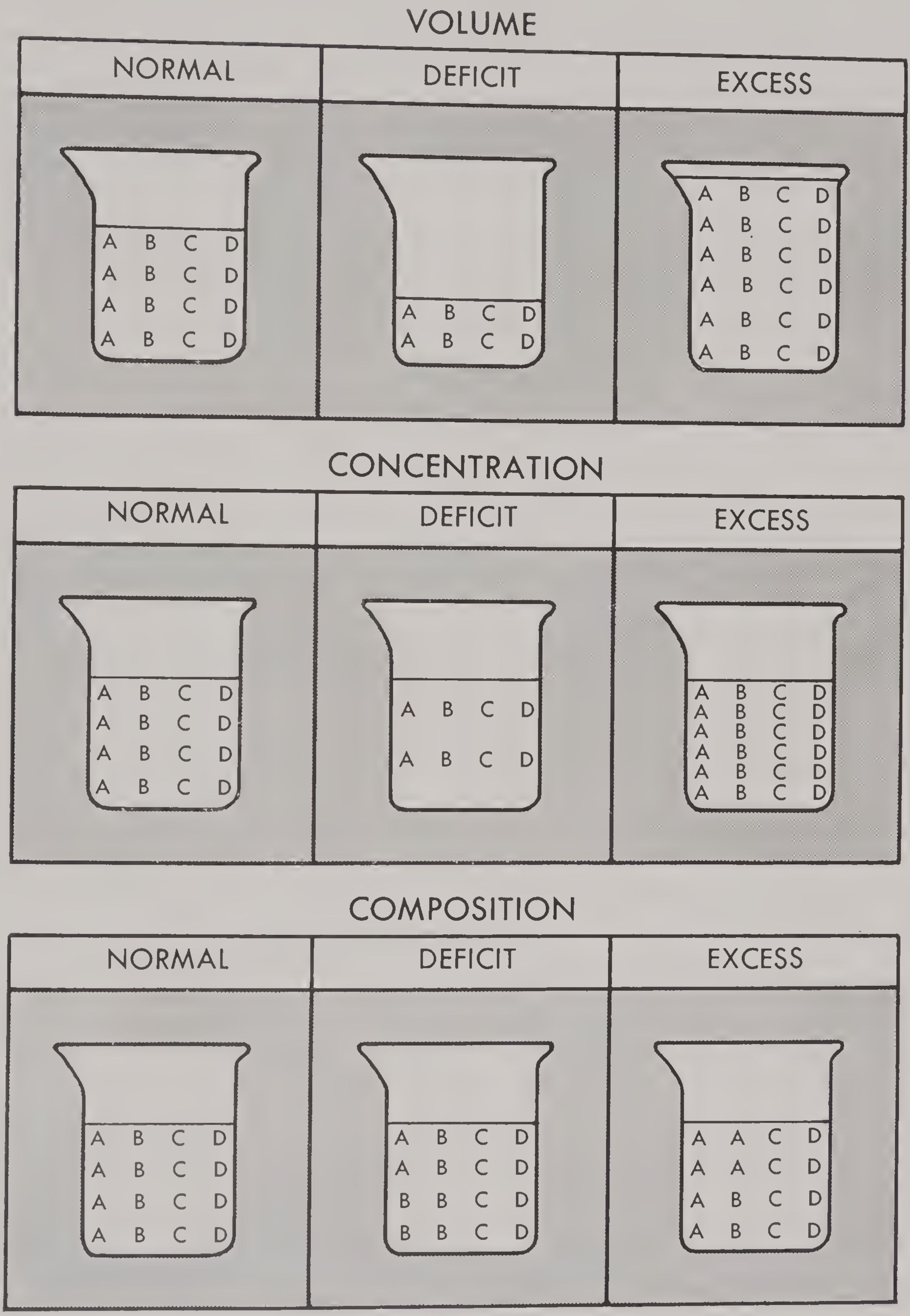


Figure 5. Beaker Analogy of Extracellular Fluid Property Changes.

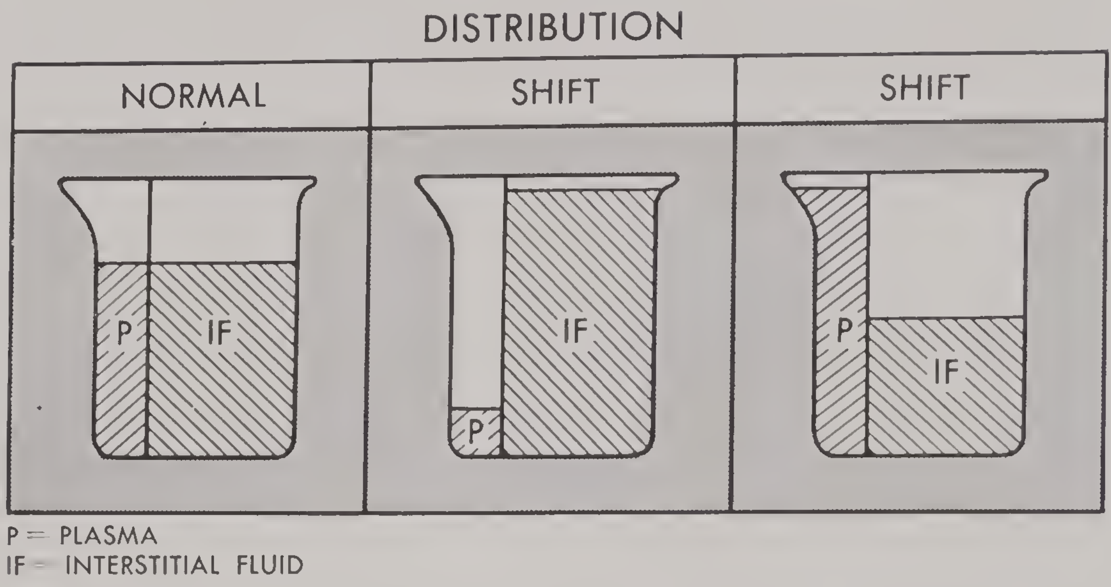


Figure 6. Beaker Analogy of Extracellular Fluid Position Changes.

hand beaker in this row is pictured as containing a normal volume of plasma. In this volume, we have listed four hypothetical electrolytes designated "A," "B," "C" and "D." Note how we can have a deficit in volume as shown in the middle beaker, or an excess in volume as shown in the right-hand beaker. In neither of these changes of extracellular fluid volume has there been a change in the concentration or composition of the contained electrolytes.

Let us view the matter of total electrolyte concentration as portrayed in the middle row of beakers. By "concentration" we mean the sum of the total number of cations and anions in milliequivalents. Without variations in either volume or composition, we can have a normal concentration of electrolytes, as demonstrated in the left-hand beaker, a deficit as shown in the middle beaker, or an excess as portrayed in the right-hand beaker.

Now let us look at the lower row. In the beaker in the left-hand position, we have shown a normal composition of plasma. In the middle beaker, we have a deficit of the electrolyte "A," and in the right-hand beaker, an excess of electrolyte "A." As a matter of fact, we can have a deficit of more than one electrolyte or we can have an excess of more than one electrolyte. We can even have combinations of deficits and excesses. These changes in composition need not be accompanied by any change in the volume or concentration of the plasma.

Any of the specific fluid imbalances just described can occur singly or in combination.

Now consider the second basic type of fluid imbalance, that which is characterized by changes in the position of the extracellular fluid. Normally, about one-fourth of the extracellular fluid exists as plasma, and about three-fourths is present as interstitial fluid (see Figure 6). The left-hand beaker shows the situation which exists when the extracellular fluid is normally distributed, one-fourth as plasma and three-fourths as interstitial fluid. The middle beaker shows the situation when, for one clinical reason or another, part of the water and electrolytes of the plasma shifts into the interstitial space. The right-hand beaker shows what happens when there is a shift of water and electrolytes from the interstitial space to the plasma. In the first case, the interstitial fluid has more than its normal share of extracellular fluid. In the second case, it is the plasma that possesses an abnormally great share.

The third basic type of fluid imbalance encompasses changes in the nutritional status of the body as a whole. It includes protein deficit, calorie deficit and vitamin deficit. These changes are necessarily slower in developing than are changes in the properties or position of the extracellular fluid, and require longer for correction. Usually more than one nutritional status change is present.

While a patient whose body fluids have been disturbed may suffer from only one fluid imbalance, most patients will manifest a combination of two or more imbalances. Just as knowledge of the clinical characteristics of individual diseases is required for the diagnosis and treatment of the patient with a combination of pathologic states, so is knowledge of the specific fluid imbalances necessary for the diagnosis and treatment of the patient with combined disturbances of the body fluids. Such knowledge, plus the facts gleaned from the clinical history, physical examination and laboratory tests, makes the diagnosis of most combined imbalances surprisingly simple.

In the next three chapters we shall examine each of the specific fluid imbalances from the standpoint of clinical history, physical examination and laboratory findings, including help from electrocardiography and roentgenography. The *principle* of management will be touched on lightly; specific therapy will be reserved for the chapters devoted to that important subject.

SUMMARY

1) Specific clinical fluid imbalances can be clearly delineated. They fall under the headings of changes in properties of extracellular fluid, changes in location of extracellular fluid and changes in nutritional status of the body.

2) While a given patient may suffer from only one imbalance, many patients manifest a combination of changes.

3) Knowledge of the clinical characteristics of specific imbalances is necessary for diagnosis of the combined imbalances so frequently seen in clinical medicine.

11

Changes in Properties of Extracellular Fluid

> *In the development of the physiology of body fluids the practicing physician has played a very active part. Initially its development was intimately related to disease: to cholera, to diabetic coma and to infantile diarrhea. . .*
>
> CARL A. MOYER

EXTRACELLULAR FLUID VOLUME CHANGES

IMBALANCE in the volume of the extracellular fluid may consist either of a deficit or of an excess. In describing changes in extracellular fluid volume, we shall outline the clinical pictures which appear when these changes are not accompanied by significant alterations in the electrolyte concentration or composition of the extracellular fluid. When concentrational or compositional changes are superimposed upon volume changes, the resulting clinical picture is a composite one, a sort of algebraic sum of the clinical pictures of the specific fluid imbalances involved. While we refer in our diagnostic classification to *extracellular fluid volume* changes, bear in mind that these may reflect and be reflected in changes in *cellular fluid volume.* For example, an extracellular fluid volume deficit in time causes a cellular fluid volume deficit. Such a combined deficit we term a *body fluid volume* deficit since it encompasses both the cellular and the extracellular fluid.

Volume Deficit

A deficit in extracellular fluid volume can be caused by an abrupt reduction in fluid intake, or by an acute loss of secretions and excretions through vomiting, severe diarrhea such as infantile diarrhea or cholera, or fistulous drainage.

On physical examination the skin feels dry. Body temperature drops because of the reduction in energy output, provided fever of infection or other extraneous conditions is not present. Evidence of central nervous system depression such as lassitude or even stupor appears. Shrinking and longitudinal wrinkling of the tongue give evidence of the reduction of the fluid volume of the tissues. Vomiting is typical of the dysfunction in gastrointestinal activity which is often present. Weight loss occurs, caused by the loss of extracellular fluid from the body. Oliguria may be found.

Pertinent laboratory findings include elevation of the hematocrit, hemoglobin and red blood cell count. The reason for these elevations lies in the fact that an extracellular fluid volume deficit involves loss of water and electrolytes but not loss of the red blood cells of the blood. The hematocrit, hemoglobin and red blood cell count values are significant only if the physician had at least a rough idea of what these values were before the onset of the extracellular fluid volume deficit.

In an extracellular fluid volume deficit, chloride is practically absent from the urine provided renal function is adequate.

The goal of therapy of extracellular fluid volume deficit is to repair the deficit by the oral or parenteral administration of a suitable solution of water and electrolytes. For the clinical picture of volume deficit see Figure 7.

Volume Excess

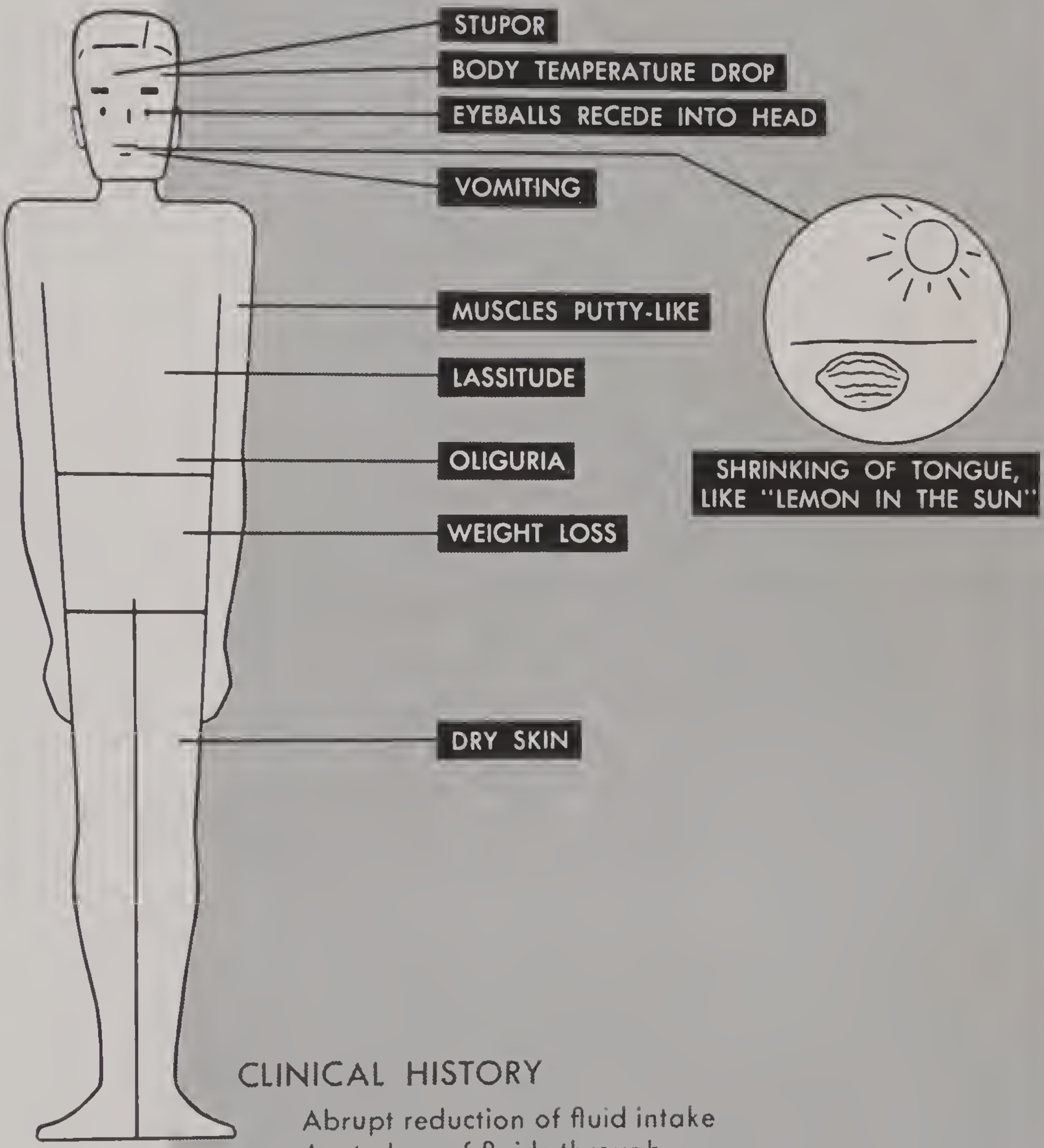
Extracellular fluid volume excess may be caused by oral or parenteral administration of excessive quantities of an isotonic solution of sodium chloride.

A chronic type of extracellular fluid volume excess may develop in congestive heart disease, chronic kidney disease or chronic liver disease with portal hypertension. Sodium is abnormally retained and, with it, water. The body homeostatic mechanisms are not functioning normally in these disorders. Malnutrition, which of itself may cause a chronic extracellular fluid volume excess, may contribute to the fluid volume excess of chronic cardiac, renal or hepatic disease.

Such clinical findings as heavy eyelids, hoarseness, shortness of breath, moist rales in the lungs, pitting edema, edema of the stomach as observed at operation, and rapid weight gain reflect the increase that occurs in tissue fluid volume. The bounding pulse reflects the increase in plasma volume.

CHANGES IN PROPERTIES OF EXTRACELLULAR FLUID

VOLUME DEFICIT



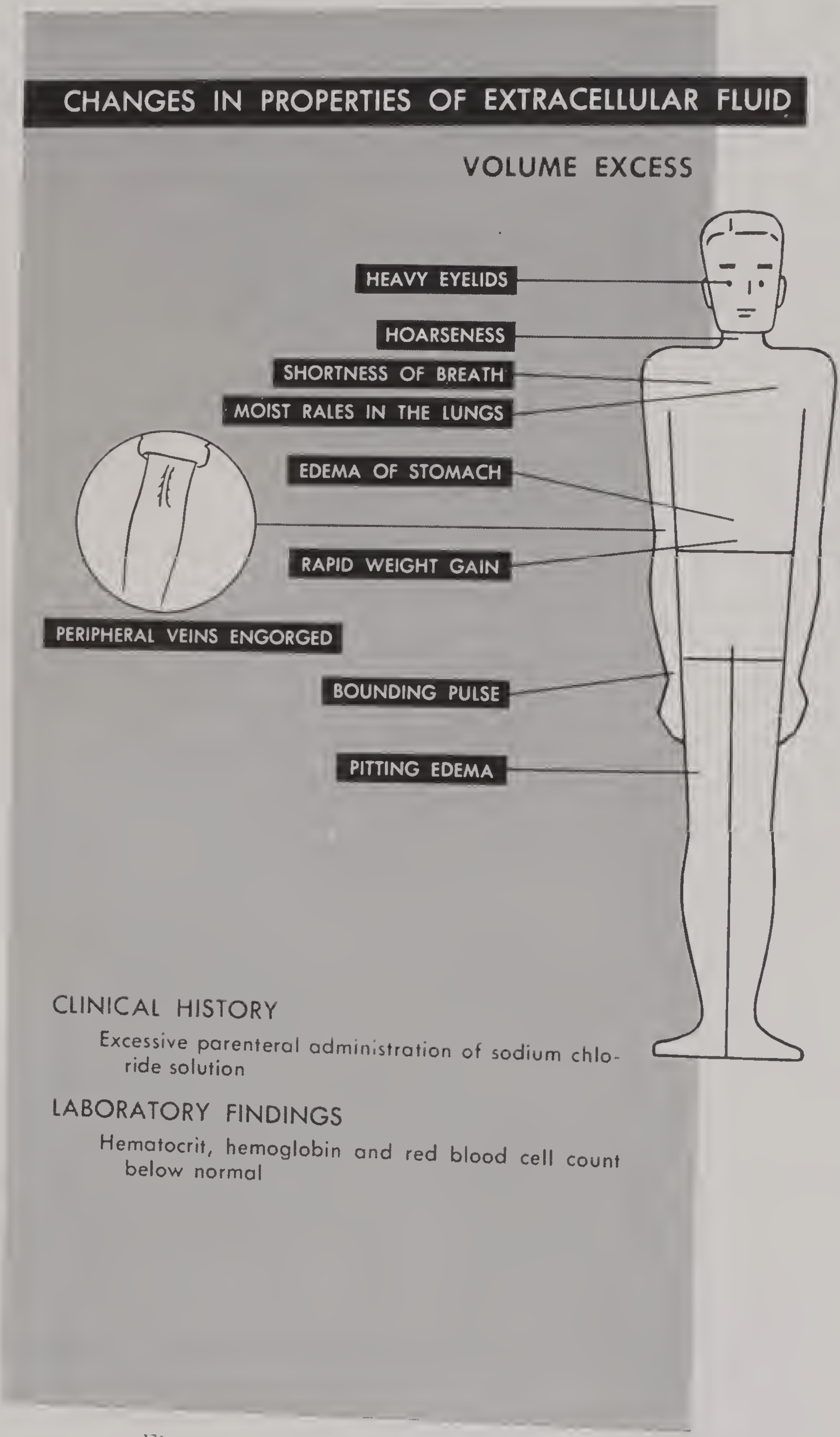
CLINICAL HISTORY

Abrupt reduction of fluid intake
Acute loss of fluids through
  vomiting
  severe diarrhea, such as infantile diarrhea or cholera
  fistulous drainage

LABORATORY FINDINGS

Hematocrit, hemoglobin and red blood cell count elevated

Figure 7. Clinical Picture of Volume Deficit.



CLINICAL HISTORY

Excessive parenteral administration of sodium chloride solution

LABORATORY FINDINGS

Hematocrit, hemoglobin and red blood cell count below normal

Figure 8. Clinical Picture of Volume Excess.

Laboratory findings include hematocrit, hemoglobin and red blood cell count values below normal. These values for the formed elements of the blood are depressed because of the dilution of the blood stream by an excess of water and electrolytes. The findings are significant only when previous values are known.

The aim of therapy of extracellular fluid volume excess is to correct the causative factors. It may be necessary to withhold all fluids for a period in acute excesses.

In fluid volume excess caused by chronic disease, various measures may be indicated including use of a low sodium diet, diuresis and correction of underlying nutritional deficits. For the clinical picture of volume excess see Figure 8.

EXTRACELLULAR FLUID CONCENTRATION CHANGES

Deficit in Total Electrolyte Concentration

Clinical causes of a deficit in total electrolyte concentration of the extracellular fluid include excessive sweating plus the drinking of water, excessive drinking of water, low sodium diet plus mercurial diuresis, repeated tap water enemas or intravenous administration of excessive amounts of carbohydrate-in-water solutions. Electrolyte concentration deficit may be seen in the patient with diarrhea whose fluid losses have been replaced with water only. Such a deficit readily occurs when a patient whose gastric or intestinal contents are being removed by suction is permitted to drink plain water. It may be caused by irrigation of gastric or intestinal suction tubes with water only. Adrenal insufficiency may also create an electrolyte concentration deficit.

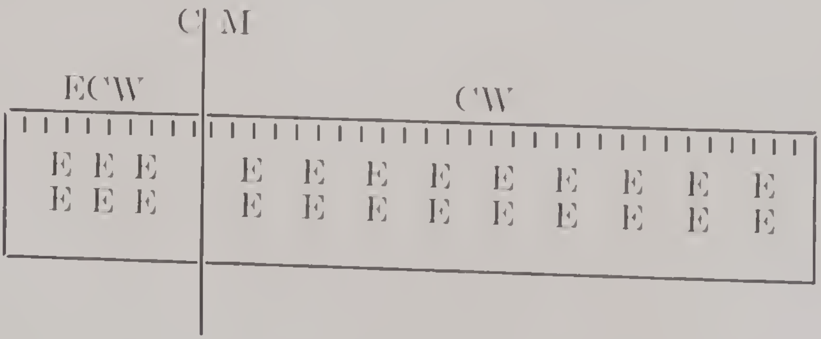
Clinically, the severity of this deficit ranges from the familiar mild heat cramps of a slight deficit to the complete collapse of a severe deficiency. Such terms as "heat cramps," "heat prostration," "water intoxication" and "low salt syndrome" represent varying clinical pictures of electrolyte concentration deficit.

The clinical findings of an acute, severe deficit in electrolyte concentration are dramatic. They are often partly attributable to an extracellular fluid volume excess which may be combined with the electrolyte concentration deficit when excessive amounts of water have been given orally or parenterally. They stand in striking contrast to the findings of a chronic, moderate electrolyte concentration deficit, with or without a fluid volume excess, which may be clinically asymptomatic.

Early clinical findings of an acute deficit in electrolyte concentration of the extracellular fluid are frequently related to disturbance of the central nervous system. These include apprehension and headache. Reflexes, at first hyperactive, may later disappear. Repeated convulsions may occur, interspersed with stuporous intervals. Convulsions are not observed in aged or debilitated patients. A related clinical finding sometimes observed is projectile vomiting, evidence of increased intracranial pressure.

Increased loss of water through excessive lacrimation, salivation and diarrhea represent compensatory actions of the body designed to reduce the fluid volume and thus increase the electrolyte concentration. Nausea and abdominal cramps are highly characteristic findings in this imbalance, indicating excessive gastrointestinal activity. Polyuria with a dilute urine may be seen initially. Later, oliguria, progressing to anuria, results from renal depression. When the fluid volume is excessive, hypertension is found; later, with progress of the imbalance, hypotension is found.

In many patients with an electrolyte concentration deficit, a visible fingerprint remains after pressure with the finger or thumb on the skin overlying the sternum. The phenomenon of fingerprinting can be explained by use of a simple diagram. The block "ECW" represents the water of extracellular fluid, and the block "CW" the water of cellular fluid. "E" represents the electrolytes of the fluids. The vertical line "CM" designates the cell membrane, which separates the extracellular fluid from the cellular fluid. When water and electrolytes are present in the two fluids in normal quantities, the following situation would obtain:



Suppose a patient develops an acute electrolyte concentration deficit of extracellular fluid because abnormal losses of water and

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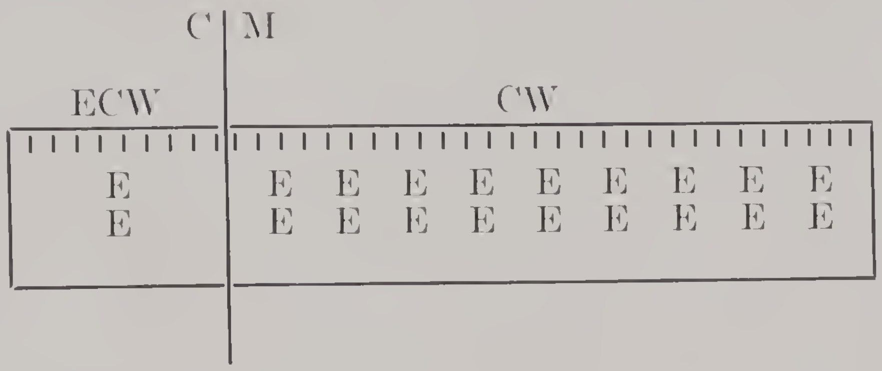
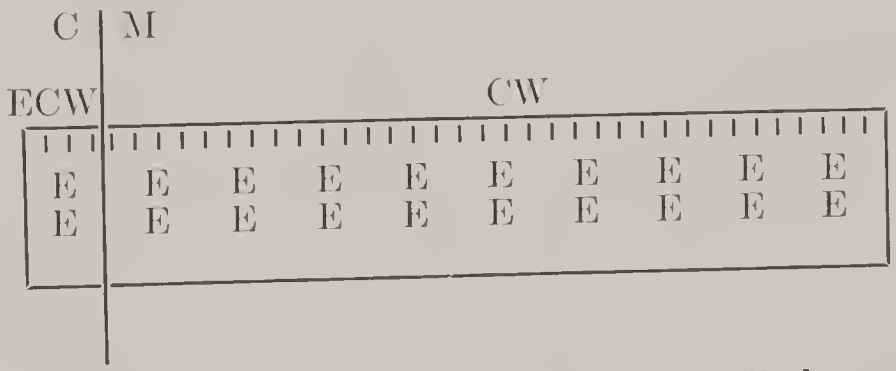
electrolytes are replaced with water only. The extracellular fluid will possess a relative excess of water as compared to the cellular fluid. In the early stages, such a condition might resemble this:



The inequality of water distribution sets into motion that "share the water" movement we call osmosis. Water passes from the extracellular fluid to the cellular fluid; the extracellular fluid volume decreases and the cellular fluid volume increases. The situation then resembles this:



As a result of the transfer of water, an abnormally large portion of the body fluids lies within the confines of the cells. The fluid volume of the extracellular fluid, both plasma and interstitial fluid, is decreased. As a result, the tissues tend to retain any shape attained by pressure deformation, that is, they become more *plastic* than normal. The clinical sign of fingerprinting is a manifestation of this increased plasticity.

The electrolyte deficit and the resultant water movement are greatly exaggerated for illustrative purposes.

Sodium is the most important cation of extracellular fluid, contributing 142 of the total 154 mEq. of cation. Chloride is the leading anion of extracellular fluid, accounting for 103 of the total 154 mEq. of anion. It is thus clear why an electrolyte concentration deficit caused by loss of electrolytes from the body results primarily from losses of sodium and chloride. Not only may a deficit in electrolyte concentration be caused by loss of water, sodium and chloride, which have been replaced by water alone, but it may result from a simple *dilution* of the extracellular fluid by excessive amounts of water.

The critical plasma sodium level which indicates a deficit in electrolyte concentration is 137 mEq. per liter. A somewhat less reliable criterion of the deficit is a sum of plasma chloride plus plasma bicarbonate less than 123 mEq. per liter. This combined value is not a reliable indication of the electrolyte concentration of the extracellular fluid when an abnormal accumulation of other anions which may displace bicarbonate or chloride exists. Thus, it is not valid when ketone bodies produce base bicarbonate deficit (metabolic acidosis), when excessive amounts of organic acids accumulate in severe renal insufficiency or when lactic acid accumulates in anoxia.

It is essential to realize that not every patient with a plasma sodium level below 137 mEq. per liter requires specific replacement therapy. Consider the patient with congestive heart failure who gradually develops an electrolyte concentration deficit in association with a fluid volume excess. Such a chronic deficit, even though indicated by a plasma sodium level as low as 130 mEq. per liter, may be unaccompanied by clinical signs and symptoms of electrolyte concentration deficit. Therapy of the deficit per se is not indicated. In contrast, the patient who has developed an electrolyte concentration deficit with a plasma sodium level of 130 mEq. per liter because sweat losses were replaced with water only, is dangerously ill. He requires prompt replacement therapy.

In planning treatment of a patient with an electrolyte concentration deficit indicated by a plasma sodium level between 130 and 137 mEq. per liter, the physician should consider the patient's clinical status. If signs and symptoms are absent, specific treatment is not necessary. If the plasma sodium level is below 130 mEq. per liter, such therapy is usually indicated even though clinical findings may be absent at the time.

Specific therapy consists of administration of a solution of sodium, the principal cation, and chloride, the principal anion, of extracellular fluid. Isotonic solution of sodium chloride is usually employed when the electrolyte concentration deficit is accompanied by a fluid volume deficit; 3% or 5% sodium chloride solution is given when the electrolyte concentration deficit is accompanied by a normal fluid volume or a fluid volume excess. For prevention and treatment of mild electrolyte concentration deficits, electrolyte tablets are useful. For the clinical picture of concentration deficit see Figure 9.

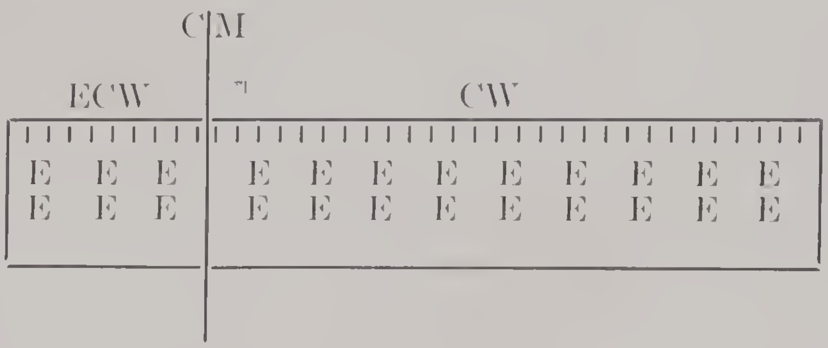
Excess in Total Electrolyte Concentration

An excess in the total electrolyte concentration of the extracellular fluid may be caused clinically by decreased water intake, especially in hot weather or febrile states, by excessive parenteral or oral administration of electrolytes, particularly hypertonic solutions, or by copious watery diarrhea. Such an excess may result from inability to swallow, such as occurs with sore throat or obstruction of the esophagus; or from unconsciousness, such as follows cerebrovascular accidents or anesthesia. Impaired renal function may also cause this imbalance. Disease interfering with the homeostatic mechanisms, such as untreated diabetes insipidus, may cause an excess in electrolyte concentration.

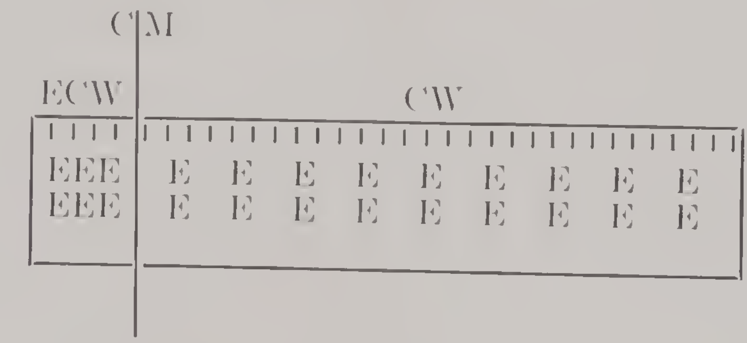
The skin is typically flushed. Physical examination reveals signs of stimulation of the central nervous system, such as excitement progressing to mania. Depressed lacrimation, depressed salivation, and thirst reflect the needs of the body for water, as do the dry, sticky mucous membranes. The tongue is rough and dry. Oliguria reveals renal depression. Other findings include hypotension, weakness and rapid heart rate. Body temperature is elevated.

The patient whose electrolyte concentration excess has resulted from abnormal losses of water, such as occur in watery diarrhea, may suffer a severe body fluid volume deficit. Paradoxically, such a patient does not usually have the dry skin and the shrunken, inelastic tissues seen in an extracellular fluid volume deficit uncomplicated by changes in electrolyte concentration. To all external appearances, he may appear quite normal with respect to his fluid stores despite a serious or even desperate body fluid volume deficit. Why is this?

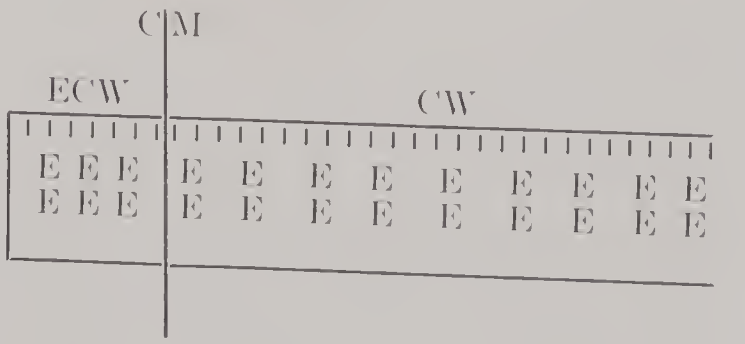
The answer to the enigma lies in a movement of water between the cellular and extracellular fluids. Let us again employ the diagram used in the previous section. It will be recalled that the block "ECW" represents the water content of extracellular fluid and "E" its electrolytes. The "CW" designates the water of cellular fluid, and "E" its contained electrolytes. "CM" is the cell membrane. The picture in normality is as follows:



Early in an electrolyte concentration excess caused by losses of water or hypotonic secretions, the extracellular fluid is deficient in its water content as compared to the cellular fluid. The diagram would appear something like this:



Osmosis occurs and water passes from the cellular to the extracellular fluid. The cellular fluid volume decreases, while the extracellular fluid volume increases, as shown by the following diagram:



It will be seen that both fluids now share what was originally an extracellular fluid volume deficit. Such a deficit is designated a *body fluid volume deficit*. Were the entire deficit suffered by the extracellular fluid, unmistakable clinical signs would be present. Since the deficit is *shared* by the extracellular and the cellular fluids, clinical signs of extracellular fluid volume deficit are minimized or even absent. A comparison of the patient's body weight with that prior to the illness will indicate a weight loss, a finding which discloses the loss of body fluid.

Both the electrolyte concentration excess and the resultant water movement are greatly exaggerated for illustrative purposes.

Laboratory findings indicative of an excess of total electrolyte concentration of extracellular fluid include a plasma sodium above 147 mEq. per liter or a sum of plasma chlorides plus plasma bicarbonate above 135 mEq. per liter. This latter value is not reliable as an indication of electrolyte concentration excess when there is an abnormal accumulation of other anions. Thus, it is not valid when excessive amounts of ketone bodies accumulate in base bicarbonate deficit (metabolic acidosis), when excessive amounts of organic acids occur in severe renal insufficiency, or when lactic acid accumulates in anoxia. The specific gravity of the urine is characteristically above 1.030 in this imbalance.

The goal of therapy is to dilute the overly concentrated extracellular fluid with an appropriate hypotonic solution of water and electrolytes. For the clinical picture of concentration excess see Figure 10.

EXTRACELLULAR FLUID COMPOSITION CHANGES

Potassium Deficit

Clinical causes of potassium deficit include conditions in which excessive amounts of potassium are lost from the body, such as: chronic pyloric obstruction with vomiting, vomiting from other causes, ulcerative colitis, fistulas of the small intestine or colon, diarrhea, especially in infants, prolonged parenteral administration of potassium-free solutions, and repeated injections of ACTH or cortisone. The deficit may be caused by endogenous overproduction of ACTH, by diabetic acidosis, and by periodic familial paralysis. Potassium deficit may develop when there is excessive production of aldosterone, a newly discovered and highly important endocrine agent which regulates sodium, potassium and chloride metabolism. Potassium deficit may also result from increased utilization of potassium by the body, during the healing phase of burns and during recovery from diabetic acidosis.

Clinical findings of potassium deficit include vomiting, anorexia and silent ileus, all indicative of dysfunction of the gastrointestinal tract. Deep reflexes are first hyperactive with neuromuscular overactivity, then disappear with the neuromuscular depression which follows. Weakness and tremor are observed. The muscles are soft, like half-filled water bottles. The intestines may be distended with gas.

The importance of the clinical considerations in the diagnosis of potassium deficit was emphasized by Darrow when he said: . . . "the diagnosis of potassium deficiency must rest largely on recognition of the conditions likely to be associated with this disturbance."

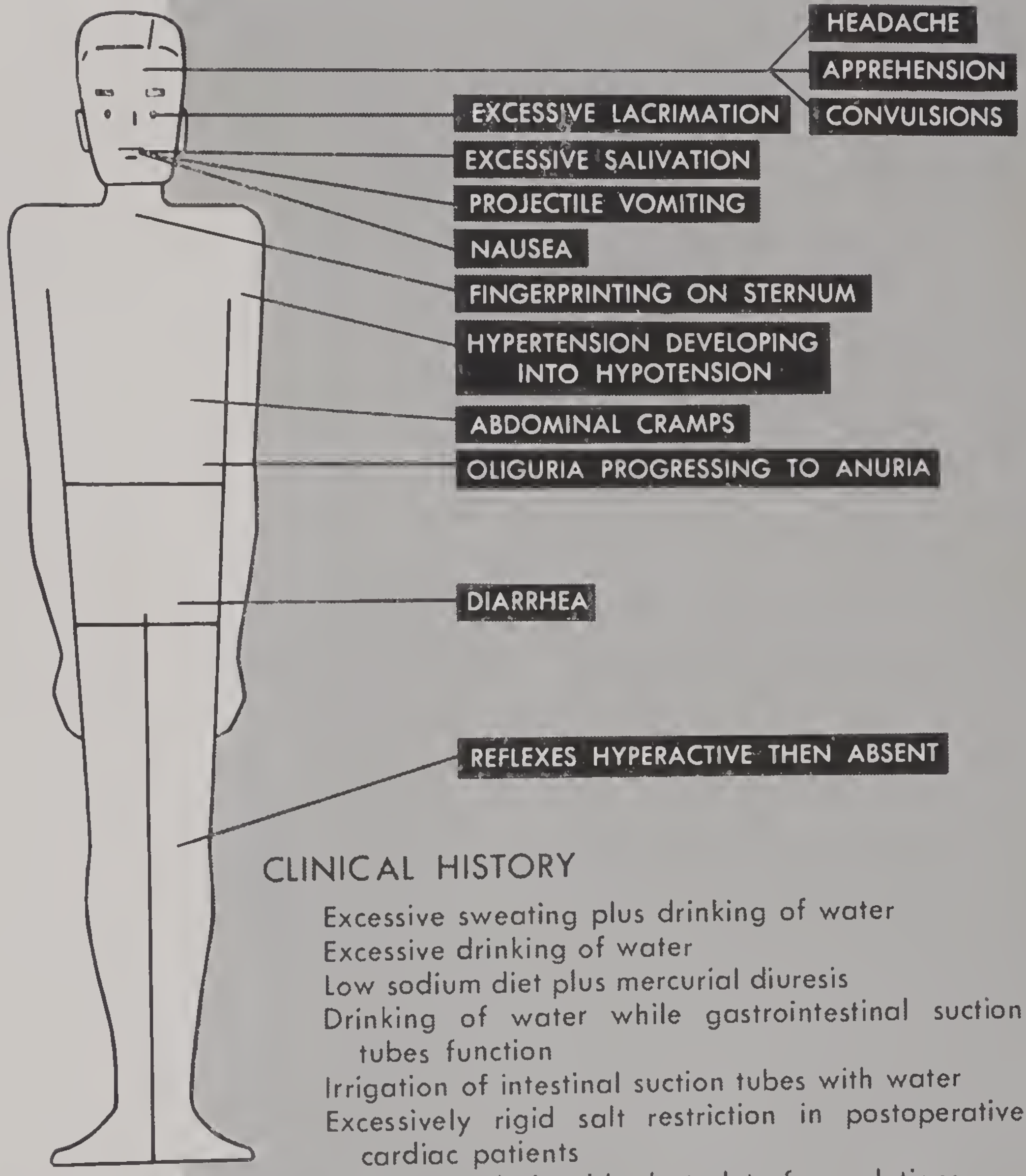
Electrocardiographic signs of low voltage, flattening of T waves and depression of the S-T segment indicate potassium deficit. A plasma potassium level below 4 mEq. per liter is confirmatory. It should be recalled that the plasma potassium level may be normal or even increased early in a *cellular* potassium deficit, since the plasma level does not always reflect the cellular stores of potassium. For example, if the patient has a severe water deficit (extracellular fluid volume deficit plus an increase in total electrolyte concentration of extracellular fluid) the plasma potassium level may be normal or elevated initially in spite of a cellular deficit.

When the patient is suffering from a base bicarbonate excess (metabolic alkalosis) which fails to respond to a chloride-containing solution, or when he has a base bicarbonate deficit (metabolic acidosis) which is resistant to a bicarbonate-containing or lactate-containing solution, potassium deficit should be considered.

Of the electrolytes which we consider in the imbalances of electrolyte composition of extracellular fluid, potassium alone is primarily a *cellular* electrolyte. Since we cannot directly assess the status of the cellular stores of potassium, we attempt to learn about them indirectly by considering what is happening to potassium in the extracellular fluid. Available laboratory tests for

CHANGES IN PROPERTIES OF EXTRACELLULAR FLUID

ELECTROLYTE CONCENTRATION DEFICIT


HEADACHE
APPREHENSION
CONVULSIONS
EXCESSIVE LACRIMATION
EXCESSIVE SALIVATION
PROJECTILE VOMITING
NAUSEA
FINGERPRINTING ON STERNUM
HYPERTENSION DEVELOPING INTO HYPOTENSION
ABDOMINAL CRAMPS
OLIGURIA PROGRESSING TO ANURIA
DIARRHEA
REFLEXES HYPERACTIVE THEN ABSENT

CLINICAL HISTORY

Excessive sweating plus drinking of water
Excessive drinking of water
Low sodium diet plus mercurial diuresis
Drinking of water while gastrointestinal suction tubes function
Irrigation of intestinal suction tubes with water
Excessively rigid salt restriction in postoperative cardiac patients
Hypodermoclysis with electrolyte-free solutions
Excessive intravenous administration of electrolyte-free solutions
Adrenal insufficiency

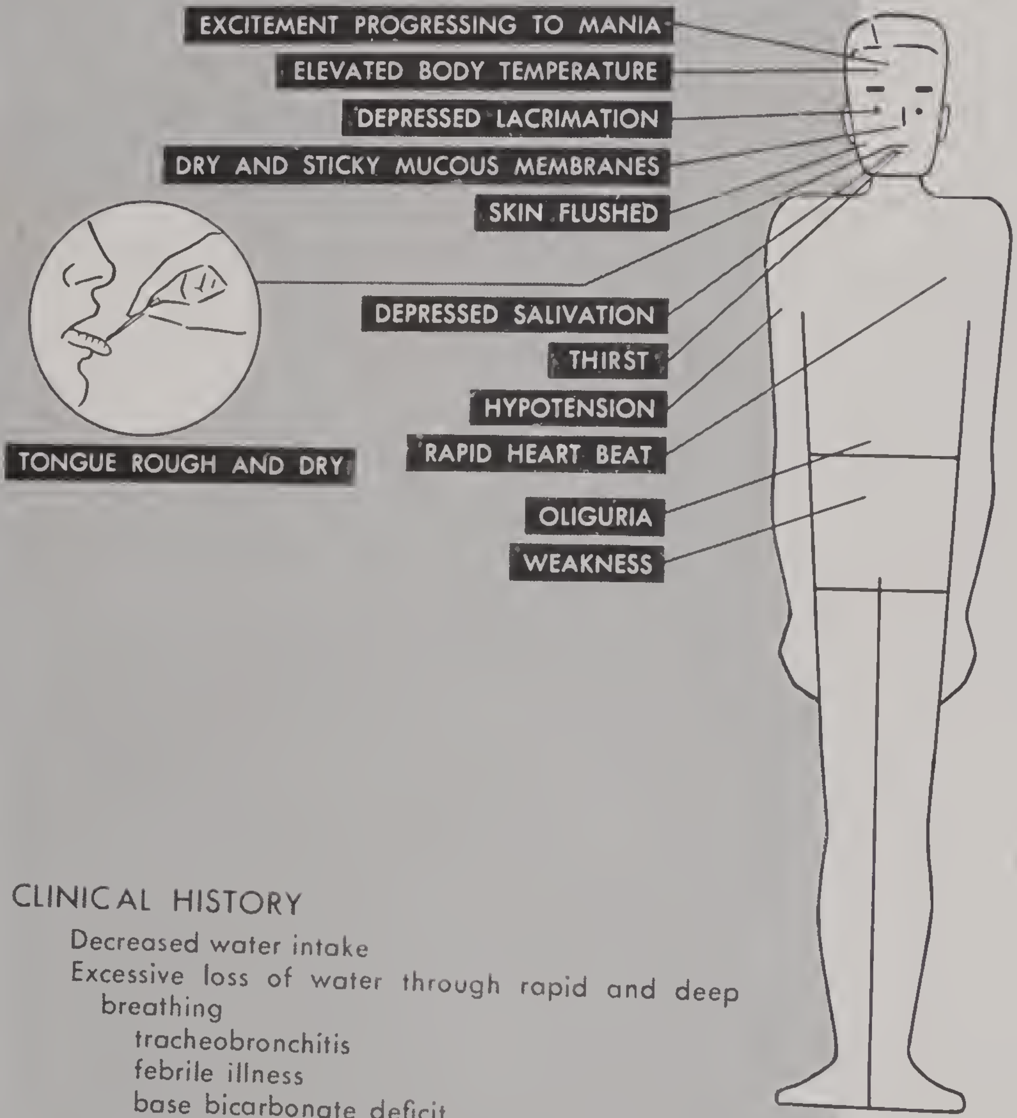
LABORATORY FINDINGS

Plasma sodium below 137 mEq./L.
Plasma chloride below 98 mEq./L.
Plasma chloride plus bicarbonate ions less than 123 mEq./L.
Specific gravity of urine below 1.010

Figure 9. Clinical Picture of Electrolyte Concentration Deficit.

CHANGES IN PROPERTIES OF EXTRACELLULAR FLUID

ELECTROLYTE CONCENTRATION EXCESS



CLINICAL HISTORY

- Decreased water intake
- Excessive loss of water through rapid and deep breathing
  - tracheobronchitis
  - febrile illness
  - base bicarbonate deficit
- Copious, watery diarrhea
- Inability to swallow
- Unconsciousness
- Impaired renal function
- Untreated diabetes insipidus

LABORATORY FINDINGS

- Plasma sodium above 147 mEq./L.
- Plasma chloride plus bicarbonate ions above 135 mEq./L.
- Specific gravity of urine above 1.030

Figure 10. Clinical Picture of Electrolyte Concentration Excess.

potassium provide us only with information concerning the potassium level of the plasma. These tests may give misleading results in certain conditions; hence, in determining the status of potassium in the cells, chief reliance must be placed on the clinical history and physical examination.

It requires several days to correct a potassium deficit because of the fact that most of the body potassium is cellular. The aim of therapy of potassium deficit is to supply needed potassium to restore the depleted cellular stores. It is preferable to give potassium by mouth if the patient can tolerate oral fluids. For the clinical picture of potassium deficit see Figure 11.

Potassium Excess

Clinical causes of potassium excess include release of excessive amounts of potassium into the extracellular fluid due to burns, scalds or crushing injuries, retention of potassium caused by severe kidney disease, excessive or too rapid parenteral administration of potassium-containing solutions or excessive oral administration of potassium-containing solutions. It also may be caused by adrenal insufficiency.

The clinical findings of potassium excess include nausea, intestinal colic and diarrhea, all of which reflect gastrointestinal hyperactivity. Oliguria progressing to anuria occurs in severe degrees of the imbalance.

The electrocardiograph shows high T waves and depressed S-T segments early. Later signs include disappearance of the P waves and heart block. The plasma potassium is typically above 5.6 mEq. per liter. This value may be normal or even decreased in *cellular* potassium excess, since the plasma potassium level does not always reflect the cellular potassium stores.

Therapy in an uncomplicated potassium excess in which the kidneys are functional consists of avoidance of additional potassium either by mouth or parenterally. When potassium excess is accompanied or caused by severe kidney disease, the physician is confronted with an extremely serious and difficult problem in therapy. For the clinical picture of potassium excess see Figure 12.

Calcium Deficit

Calcium deficit of the extracellular fluid may occur in such clinical conditions as acute pancreatitis, massive infections of subcutaneous tissues, burns during the slough and early granulation stages, and generalized peritonitis. Apparently, in these conditions large amounts of calcium are immobilized in the diseased tissue and exudates. The deficit may occur also when parathyroid tissue is inadvertently removed during surgical operation on the thyroid gland. Such surgical extirpation of parathyroid tissue destroys the controlling mechanism for maintenance of the normal plasma calcium level. There is, likewise, interference with the control of the plasma calcium in primary hypoparathyroidism, pseudohypoparathyroidism, and vitamin D resistant rickets. Chronic renal disease may be associated with calcium deficit.

Calcium deficit may result from loss of calcium in the fatty stools of steatorrhea, such as occurs in sprue and pancreatic deficiency, in intestinal shunt operations and in fluoride poisoning.

It is the ionized portion of the plasma calcium which is important from the standpoint of normal neuromuscular function. In health this ionized fraction equals about one-half of the total plasma calcium. When the extracellular fluid becomes relatively acidic in reaction, the ionized calcium increases at the expense of the nonionized fraction. It is for this reason that a patient whose *total plasma calcium* is *low* may have a *normal ionized plasma calcium* level in the presence of acidosis.

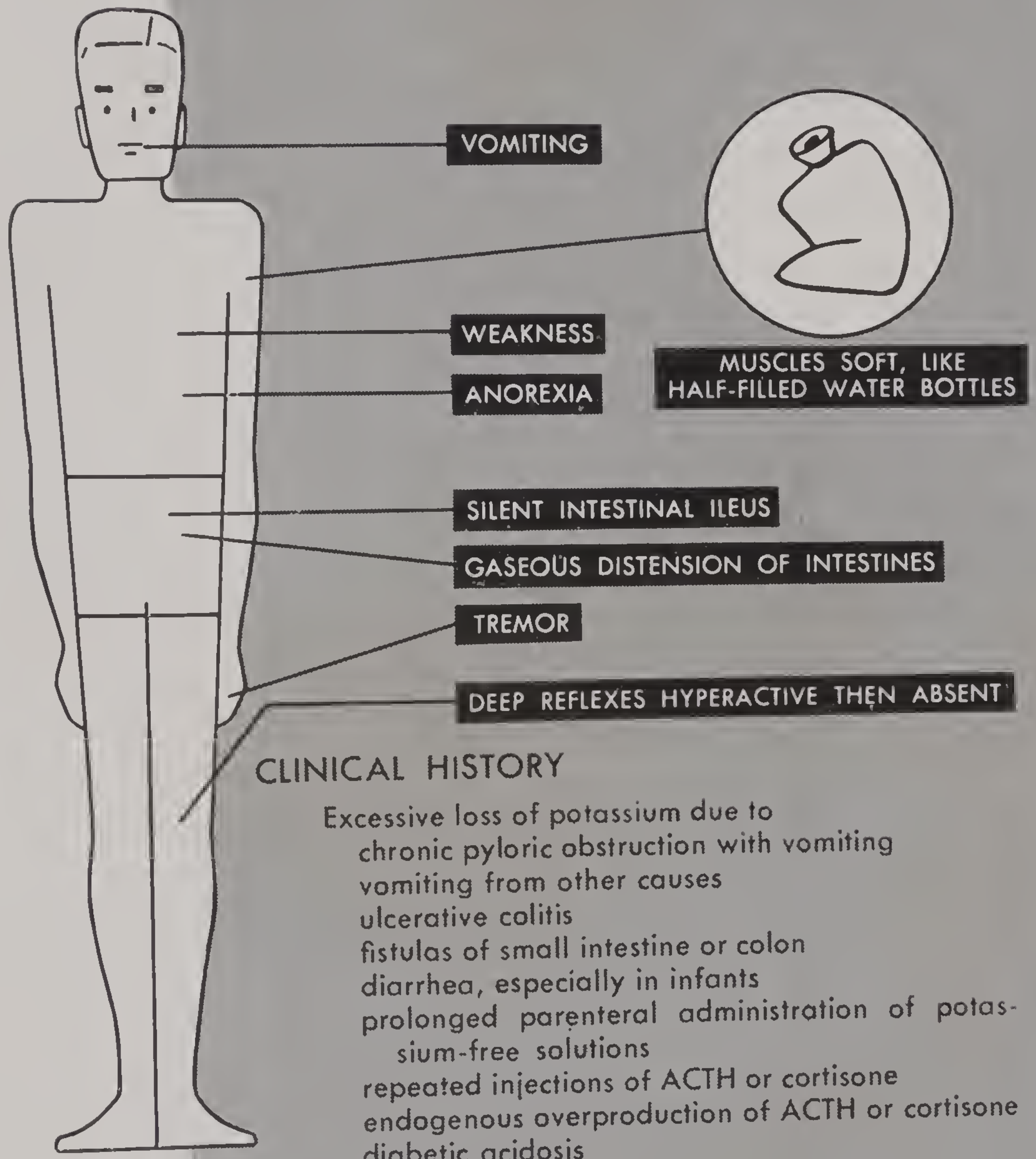
Should the extracellular fluid become basic in reaction, the ionized portion of the calcium decreases. Rapid correction or overcorrection of acidosis, therefore, may cause symptoms of calcium deficit such as tetany.

Calcium deficit may be caused by the formation of a bound form of calcium when excessive amounts of citrated blood are given.

The clinical findings of calcium deficit include tetany, laryngeal stridor, tingling of the fingers and of the circumoral region, and abdominal cramps. The physician may find numbness of the extremities, a carpopedal attitude of the hands, muscular cramps

CHANGES IN PROPERTIES OF EXTRACELLULAR FLUID

POTASSIUM DEFICIT


VOMITING
MUSCLES SOFT, LIKE HALF-FILLED WATER BOTTLES
WEAKNESS
ANOREXIA
SILENT INTESTINAL ILEUS
GASEOUS DISTENSION OF INTESTINES
TREMOR
DEEP REFLEXES HYPERACTIVE THEN ABSENT

CLINICAL HISTORY

- Excessive loss of potassium due to
  - chronic pyloric obstruction with vomiting
  - vomiting from other causes
  - ulcerative colitis
  - fistulas of small intestine or colon
  - diarrhea, especially in infants
  - prolonged parenteral administration of potassium-free solutions
  - repeated injections of ACTH or cortisone
  - endogenous overproduction of ACTH or cortisone
  - diabetic acidosis
  - periodic familial paralysis
- Increased utilization of potassium
  - healing phase of burns
  - recovery from diabetic acidosis

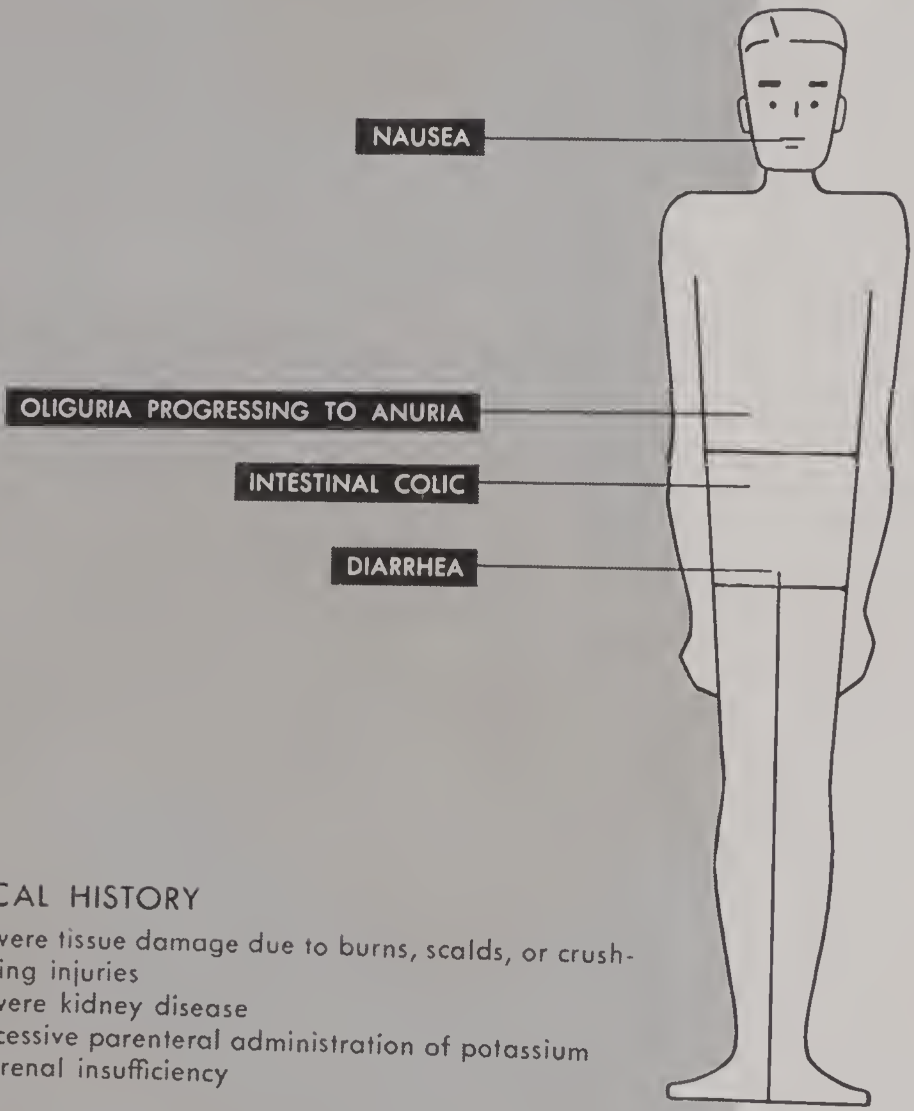
LABORATORY FINDINGS

- Electrocardiograph shows low voltage, flattening of T waves. Depression of S-T segment
- Plasma potassium below 4 mEq./L.
- Base bicarbonate excess resistant to chloride administration, or base bicarbonate deficit resistant to lactate or to bicarbonate administration

Figure 11. Clinical Picture of Potassium Deficit.

CHANGES IN PROPERTIES OF EXTRACELLULAR FLUID

POTASSIUM EXCESS



CLINICAL HISTORY

- Severe tissue damage due to burns, scalds, or crushing injuries
- Severe kidney disease
- Excessive parenteral administration of potassium
- Adrenal insufficiency

LABORATORY FINDINGS

- Electrocardiograph shows high T waves and depressed S-T segments. Later signs are disappearance of P waves and heart block
- Plasma potassium above 5.6 mEq./L.

Figure 12. Clinical Picture of Potassium Excess.

and hyperactivity of the reflexes, as demonstrated by Chvostek's sign, elicited by tapping the facial nerve.

The electrocardiographic tracing in calcium deficit reveals a prolongation of the Q-T interval. Among the laboratory findings the plasma calcium is ordinarily below 4.5 mEq. per liter. This level includes both the ionized and the nonionized calcium.

The proportion of the total calcium which exists in the ionized form is determined by the level of the plasma proteins, especially the plasma albumin. As the plasma proteins rise, the proportion of ionized calcium decreases. As the plasma proteins decrease, the proportion of ionized calcium rises.

The decreased plasma calcium level present in hypoparathyroidism is often associated with a plasma phosphorus above the normal level of 2.6 mEq. per liter.

Urinary calcium excretion is decreased when the plasma calcium level is decreased. The amount of calcium excreted in the urine can be roughly estimated by use of the Sulkowitch reagent, a buffered solution of oxalates. When this reagent is added to urine an immediate precipitate of calcium oxalate is produced. The density of the precipitate varies with the amount of calcium present in the urine. If no precipitate forms when the Sulkowitch reagent is added to urine, the plasma calcium level is probably below normal.

Therapy of calcium deficit includes the intravenous administration of calcium gluconate if the deficit is acute. If the deficit is not acute, calcium lactate may be given by mouth along with a diet containing generous amounts of calcium-rich foods. For the clinical picture of calcium deficit see Figure 13.

Calcium Excess

Calcium excess of the extracellular fluid may result from overactivity of the parathyroid glands. It may be caused by neoplasms of these organs. Other causes of calcium excess include multiple myeloma, multiple fractures, prolonged body immobilization and excessive administration of vitamin D. The latter may occur when large doses of vitamin D are used in the treatment of arthritis.

The clinical findings of calcium excess are caused by the high level of calcium in the plasma, or by the excessive removal of calcium from the bones due to the overactivity of the parathyroid glands. The findings include hypotonicity of the muscles, deep bony pain, cavitation of the bones, and renal concretions.

X-ray examination may reveal generalized osteoporosis, widespread cavitation of the bones or radiopaque urinary stones. The urinary output of calcium is greatly increased, as shown by a heavy precipitate when the Sulkowitch test is performed. The plasma calcium level is usually above 5.8 mEq. per liter.

Therapy of calcium excess aims at correction of the cause. If other fluid imbalances are being treated, only parenteral solutions free of calcium should be employed. For the clinical picture of calcium excess see Figure 14.

Introduction to Acid-Base Imbalances

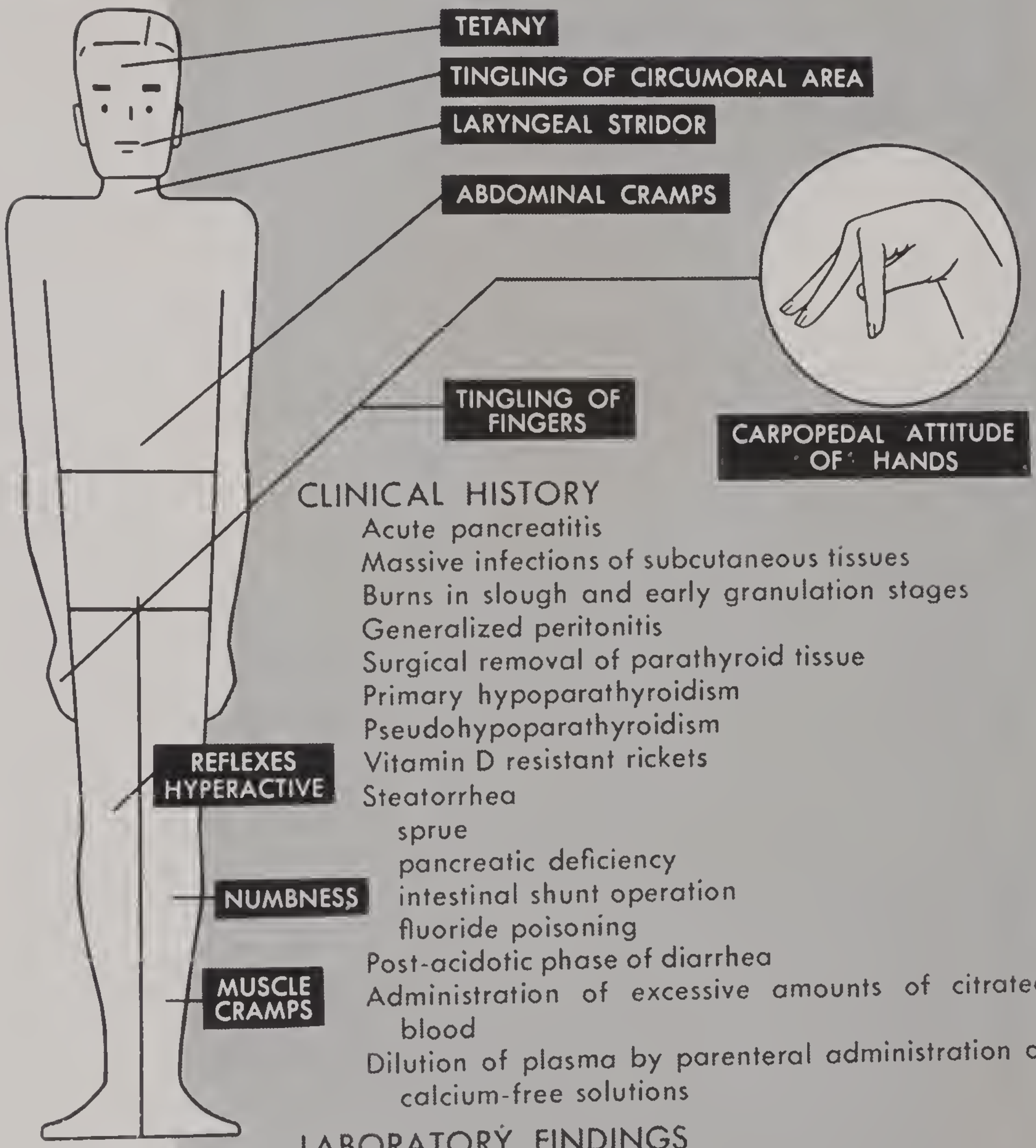
From the standpoint of the clinician, the several acid-base imbalances may be regarded as resulting from base bicarbonate deficit, base bicarbonate excess, carbonic acid deficit or carbonic acid excess. *The state of acid-base balance or imbalance of the extracellular fluid depends primarily upon the relative quantities of carbonic acid and base bicarbonate present in the extracellular fluid.*

We shall purposely disregard the other chemical systems which play a minor role in the regulation of the acid-base state. These include the monosodium phosphate:disodium phosphate system and the plasma protein system of the extracellular fluid; and the monopotassium phosphate:dipotassium phosphate system and the oxyhemoglobin:reduced hemoglobin system of the red blood cells.

In arriving at a clinical understanding of the acid-base imbalances, let us first recall that the degree of acidity or alkalinity of a fluid is determined by the concentration of hydrogen (H^+) ions present. The greater the concentration of hydrogen ions, the higher the acidity. The lower the concentration of hydrogen ions, the lower the acidity. Now the concentration of hydrogen ions in extracellular fluid is extremely small. The actual value lies between .0000001 and .00000001 Gm. per liter of extracellular fluid. To report hydrogen ions employing such infinitesimal figures would be highly inconvenient. So it was for this reason that

CHANGES IN PROPERTIES OF EXTRACELLULAR FLUID

CALCIUM DEFICIT



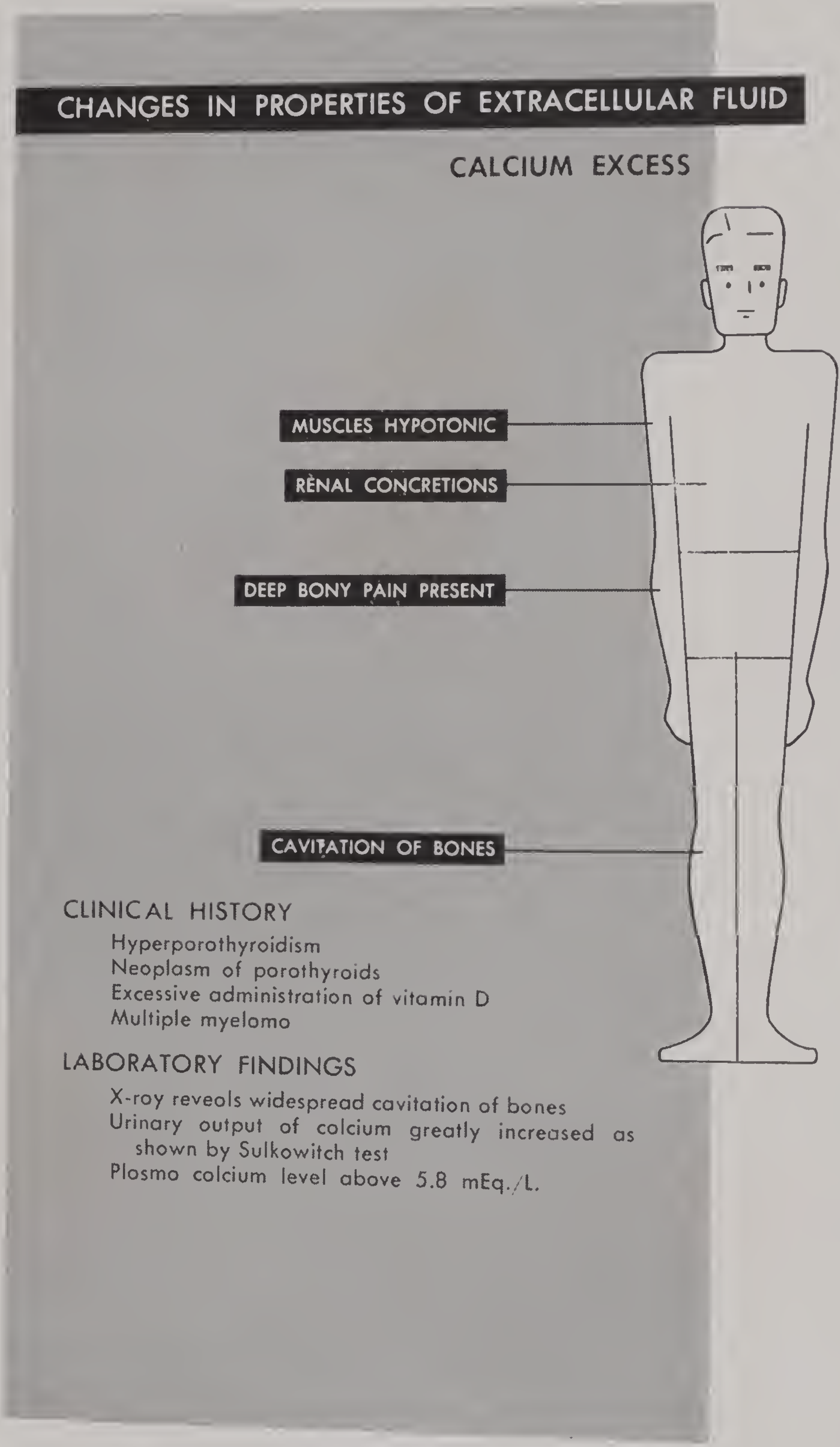
CLINICAL HISTORY

- Acute pancreatitis
- Massive infections of subcutaneous tissues
- Burns in slough and early granulation stages
- Generalized peritonitis
- Surgical removal of parathyroid tissue
- Primary hypoparathyroidism
- Pseudohypoparathyroidism
- Vitamin D resistant rickets
- Steatorrhea
  - sprue
  - pancreatic deficiency
  - intestinal shunt operation
  - fluoride poisoning
- Post-acidotic phase of diarrhea
- Administration of excessive amounts of citrated blood
- Dilution of plasma by parenteral administration of calcium-free solutions

LABORATORY FINDINGS

- Electrocardiograph shows prolongation of Q-T interval
- Plasma calcium below 4.5 mEq./L.
- Plasma phosphorus may be above 2.6 mEq./L. if hypoparathyroidism is present
- If plasma protein, especially albumin, is depressed, total plasma calcium can be lower than normal with ionized calcium still within normal bounds. Plasma proteins determine portion of total plasma calcium which is ionized
- Sulkowitch test on urine shows definite precipitation when plasma calcium concentration is normal

Figure 13. Clinical Picture of Calcium Deficit.



CLINICAL HISTORY

- Hyperparathyroidism
- Neoplasm of parathyroids
- Excessive administration of vitamin D
- Multiple myeloma

LABORATORY FINDINGS

- X-ray reveals widespread cavitation of bones
- Urinary output of calcium greatly increased as shown by Sulkowitch test
- Plasma calcium level above 5.8 mEq./L.

Figure 14. Clinical Picture of Calcium Excess.

the pH method of expressing hydrogen ion values was devised.

The pH may be defined as the "negative logarithm of the hydrogen ion concentration." Since the logarithm is a negative one, the pH value decreases as the hydrogen ion concentration and, hence, acidity, of a fluid increases, and the pH value increases as the alkalinity increases. Each pH value represents ten times as many hydrogen ions as the next larger numerical value. For example, a pH of 7 represents ten times as many hydrogen ions as a pH of 8. A pH of 6 represents ten times as many hydrogen ions as a pH of 7, 100 times as many as a pH of 8, and so on.

The extracellular fluid of the body is normally maintained within the narrow range of pH 7.35 to 7.45. As long as the pH lies between these two values, the patient is in acid-base balance. When the pH drops below 7.35, acidosis is said to be present. We use the term *acidemia* as a synonym for acidosis. When the pH of the extracellular fluid rises above 7.45, then alkalosis is present. We employ the term *alkalemia* as synonymous with alkalosis. Death occurs below a pH of approximately 6.8 and above a pH of about 7.8 (see plate VIII).

The normal pH of extracellular fluid is considerably more alkaline than the pH of water, which is about 7. If one considers the water pH of 7 as neutral, the normal pH of extracellular fluid lies in the range of alkalinity. If one regards the pH of body fluid as neutral, then water is an acidic substance indeed. If one's body fluid had the pH of distilled water, one would be near death from acidosis.

What is it that determines the pH of the extracellular fluid? *The important determinant is the ratio of carbonic acid to base bicarbonate.* Normally, there are 1.33 mEq. of carbonic acid and 27 mEq. of base bicarbonate in each liter of extracellular fluid. This is roughly a ratio of 1 part of carbonic acid to 20 parts of base bicarbonate. As long as this *ratio* is maintained, the pH of the extracellular fluid will lie within the normal range of 7.35 to 7.45.

If the carbonic acid and the base bicarbonate of the fluid are both divided by two or both multiplied by two, the ratio remains 1 to 20, the pH of the extracellular fluid remains between 7.35 and 7.45, and the patient remains in a state of acid-base balance. If either the carbonic acid or base bicarbonate is increased or decreased so that the ratio of 1 to 20 no longer holds, an acid-base imbalance results.

We like to visualize this state of acid-base balance of the extracellular fluid (and of the patient) as a teeter-totter with 1 part of carbonic acid resting on the left side and 20 parts of base bicarbonate resting on the right side. As long as the ratio of 1 to 20 is maintained, the teeter-totter remains level, the pH remains within the normal limits of 7.35 to 7.45, and the patient remains in acid-base balance. If the teeter-totter is tipped to the left, the pH drops below 7.35 and acidosis occurs. If the teeter-totter tips to the right, the pH rises above 7.45 and alkalosis is present.

Two general types of disturbances tilt our mythical teeter-totter. *Metabolic* or *systemic* disturbances affect the base bicarbonate or *right side* of the teeter-totter. If such a disturbance removes base bicarbonate, a base bicarbonate deficit occurs and the teeter-totter tips to the left. Acidosis is present. If a disturbance causes an increase of base bicarbonate, a base bicarbonate excess occurs and the teeter-totter tips to the right. Alkalosis is present. Since these imbalances are caused by metabolic conditions, they are referred to as *metabolic* acidosis or *metabolic* alkalosis.

Respiratory disturbances affect the carbonic acid or *left side* of the teeter-totter. If such a disturbance decreases the amount of carbonic acid present, a carbonic acid deficit occurs. Alkalosis is present. If the pathologic state results in an increase of carbonic acid, a carbonic acid excess results. Acidosis is present. Since these imbalances are caused by respiratory conditions, they are referred to as *respiratory* alkalosis or *respiratory* acidosis.

Now let us examine the several acid-base imbalances from the standpoint of their clinical causes, clinical findings and the principle of therapy.

Base Bicarbonate Deficit (Metabolic Acidosis)

Picture the acid-base teeter-totter of the patient as level prior to the onset of base bicarbonate deficit (see Figures 15 and 16). There is 1 part of carbonic acid to 20 parts of base bicarbonate. The pH of the body fluids lies within the normal range.

Any clinical event which causes the production of excessive amounts of acidic materials such as ketone, chloride or organic acid ions may upset the balance. Diarrhea, particularly in infants, uncontrolled diabetes mellitus, starvation, severe infectious disease or a ketogenic diet causes production of ketone ions; they result from the metabolism of the body fats which occurs after the available body stores of glycogen have been depleted. Excessive parenteral administration of a solution of sodium chloride floods the extracellular fluid with chloride ions; excessive ingestion of ammonium chloride has the same effect. Renal insufficiency causes retention of organic acid ions.

Whatever the reason for their presence, ketone, chloride or organic acid ions replace bicarbonate ions and tend to tilt the teeter-totter to the left. If enough acidic ions are present, tilting occurs. The pH of the extracellular fluid drops below 7.35. Acidosis is present.

The base bicarbonate deficit of metabolic acidosis is said to be *primary* since it is the immediate cause of the disturbance of acid-base balance.

Immediately the body begins compensatory action. The lungs blow off carbonic acid, as carbon dioxide, in an effort to lighten the carbonic acid side of the teeter-totter and thus restore balance. This action of the lungs is responsible for the hyperactive "Kussmaul" type of respiration which characterizes metabolic acidosis.

In a mild base bicarbonate deficit, only shortness of breath may occur. In moderate and severe metabolic acidosis, breathing becomes deep and periodic in older children and adults, deep and rapid in infants and young children. In some instances of severe acidosis, breathing may actually be hypoactive. Hyperactive breathing is usually not present in very young infants with metabolic acidosis.

Along with symptoms caused by the body compensatory action, other symptoms occur: weakness, disorientation and stupor progressing to coma. The kidneys assist in compensation. They conserve base bicarbonate while excreting hydrogen ions and nonbicarbonate anions. The urine becomes acid.

The laboratory findings in base bicarbonate deficit include an acid urine with pH below 6.0. The urine may, however, be alkaline in acidosis when certain conditions which are listed in chapter 15 are present. Nitrazine® paper gives a pH reading readily when the color comparison chart provided with the papers is used. The plasma pH will be found to be below 7.35. The plasma bicarbonate (HCO_3) is below 25 mEq. per liter in adults and below 20 mEq. per liter in children. The plasma bicarbonate invaribly reflects the level of base bicarbonate. As will be seen, it does not indicate whether acidosis or alkalosis is present. Of itself, it gives no clue to the pH of the extracellular fluid.

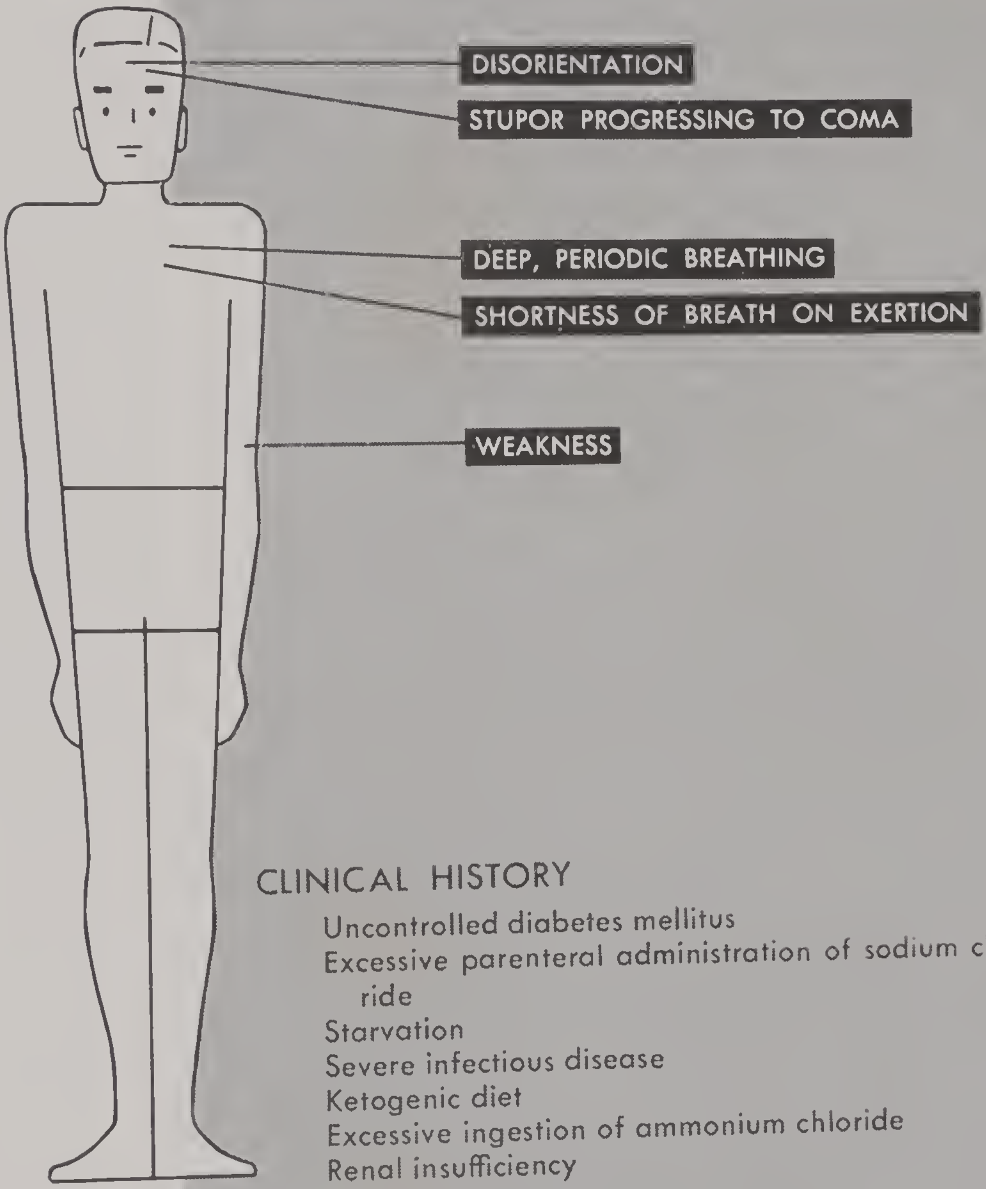
The goal of therapy of base bicarbonate deficit is to restore the level of base bicarbonate of the extracellular fluid. This can be done by administering a solution of sodium bicarbonate. Since this may be hazardous, solutions containing sodium lactate are usually employed. Lactate ions are metabolized in the liver and are replaced by bicarbonate ions, which restore the base bicarbonate side of the teeter-totter.

Of great importance in management of base bicarbonate deficit is the provision of a generous amount of fluid to repair the fluid volume deficit which is invariably present. Carbohydrate is administered as part of the parenteral solution to provide a fat-free source of enegry.

A 0.9% solution of sodium chloride, also designated "normal saline" or "physiological solution of sodium chloride," has been employed in the management of base bicarbonate deficit as well

CHANGES IN PROPERTIES OF EXTRACELLULAR FLUID

BASE BICARBONATE DEFICIT (Metabolic Acidosis)



CLINICAL HISTORY

Uncontrolled diabetes mellitus
Excessive parenteral administration of sodium chloride
Starvation
Severe infectious disease
Ketogenic diet
Excessive ingestion of ammonium chloride
Renal insufficiency

LABORATORY FINDINGS

Urine pH below 6.0
  Nitrazine® paper gives pH reading by use of pH color comparison chart provided with papers
Plasma pH below 7.35
Plasma bicarbonate below 25 mEq./L. in adults and below 20 mEq./L. in children

Figure 15. Clinical Picture of Base Bicarbonate Deficit.

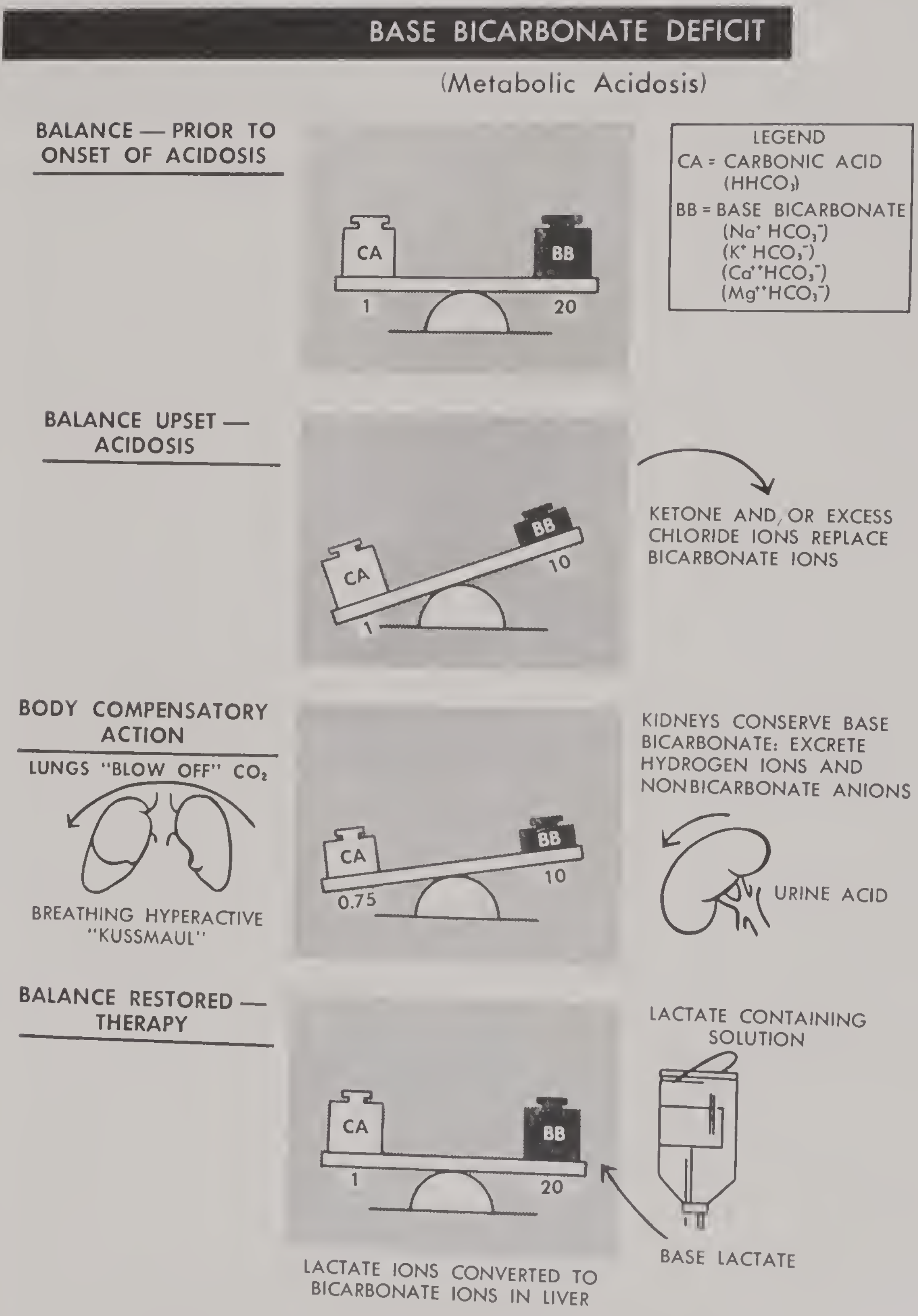

BASE BICARBONATE DEFICIT
(Metabolic Acidosis)
BALANCE — PRIOR TO ONSET OF ACIDOSIS
CA
BB
1
20
LEGEND
CA = CARBONIC ACID ($HHCO_3$)
BB = BASE BICARBONATE
($Na^+ HCO_3^-$)
($K^+ HCO_3^-$)
($Ca^{++} HCO_3^-$)
($Mg^{++} HCO_3^-$)
BALANCE UPSET — ACIDOSIS
CA
BB
1
10
KETONE AND/OR EXCESS CHLORIDE IONS REPLACE BICARBONATE IONS
BODY COMPENSATORY ACTION
LUNGS "BLOW OFF" CO_2
BREATHING HYPERACTIVE "KUSSMAUL"
CA
BB
0.75
10
KIDNEYS CONSERVE BASE BICARBONATE: EXCRETE HYDROGEN IONS AND NONBICARBONATE ANIONS
URINE ACID
BALANCE RESTORED — THERAPY
CA
BB
1
20
LACTATE CONTAINING SOLUTION
BASE LACTATE
LACTATE IONS CONVERTED TO BICARBONATE IONS IN LIVER

Figure 16. Base Bicarbonate Deficit Teeter-Totter.

as of many other fluid imbalances. Such a solution of sodium chloride, which we prefer to call "isotonic solution of sodium chloride," is actually contraindicated for many of the conditions in which it has been employed. This is particularly true in the case of base bicarbonate deficit (metabolic acidosis).

Although called "physiological," this solution is actually *unphysiological.* The reason is this: extracellular fluid contains 142 mEq. of sodium per liter, but an isotonic solution of sodium chloride contains 154 mEq. of sodium per liter. The sodium content of isotonic solution of sodium chloride is thus 12 mEq. per liter above the sodium content of extracellular fluid. This is not ideal, but it is not definitely harmful. The chloride of extracellular fluid is 103 mEq. per liter. The chloride of isotonic solution of sodium chloride is 154 mEq. per liter, an excess of 51 mEq. of chloride per liter. Therefore, isotonic solution of sodium chloride forces an excess of 51 mEq. of chloride upon the body for each liter of the solution given.

Now the kidneys must maintain the extracellular fluid constant in electrolyte composition. Thus, an additional work load is imposed upon the kidneys when such a solution is given, for they must excrete the excess of 51 mEq. of chloride for each liter given. If the kidneys are already under stress, the imposition of this additional load is especially serious. In some situations it may actually encourage development of base bicarbonate deficit (metabolic acidosis), since the excess chloride tends to decrease the relative amount of base bicarbonate in the extracellular fluid and, hence, to disturb the carbonic acid:base bicarbonate ratio.

Base Bicarbonate Excess (Metabolic Alkalosis)

Before the onset of metabolic alkalosis, our acid-base teeter-totter is in the level position (see Figures 17 and 18). An excess of base bicarbonate is most frequently caused by loss of chloride and potassium through vomiting, such as occurs in pyloric obstruction, high intestinal obstruction or pernicious vomiting of pregnancy. X-ray therapy or other conditions which encourage potassium loss may cause base bicarbonate excess. These include hyperadrenalism, which may result from overactivity of the anterior pituitary gland, an adrenal tumor, an overdosage of ACTH or an overdosage of adrenal corticoids such as compounds E or F. Prolonged intravenous alimentation with potassium-free solutions may have the same result.

Loss of chloride causes a compensatory increase of base bicarbonate. In this connection recall that the total height of the anion column of electrolytes must always equal the total height of the cation column. Loss of potassium encourages base bicarbonate excess in a manner that is not entirely understood. When the patient ingests excessive amounts of sodium bicarbonate, as in improperly controlled alkali therapy of peptic ulcer, the base bicarbonate can be increased excessively.

When the amount of base bicarbonate in the extracellular fluid becomes excessive for any cause, the teeter-totter is tipped to the right. The pH rises above 7.45. Alkalosis is present.

The base bicarbonate excess of metabolic alkalosis is said to be *primary* since it is the proximate cause of the disturbance of the acid-base imbalance.

Body compensatory action immediately comes into play. The lungs hold back carbon dioxide in an effort to build up the carbonic acid side of the teeter-totter and restore balance. This results in the suppression of breathing sometimes noticed as a symptom of base bicarbonate excess. The kidneys excrete bicarbonate ions, and retain hydrogen ions and nonbicarbonate anions in an effort to restore the normal carbonic acid:base bicarbonate ratio. It is for this reason that the urine is found to be alkaline.

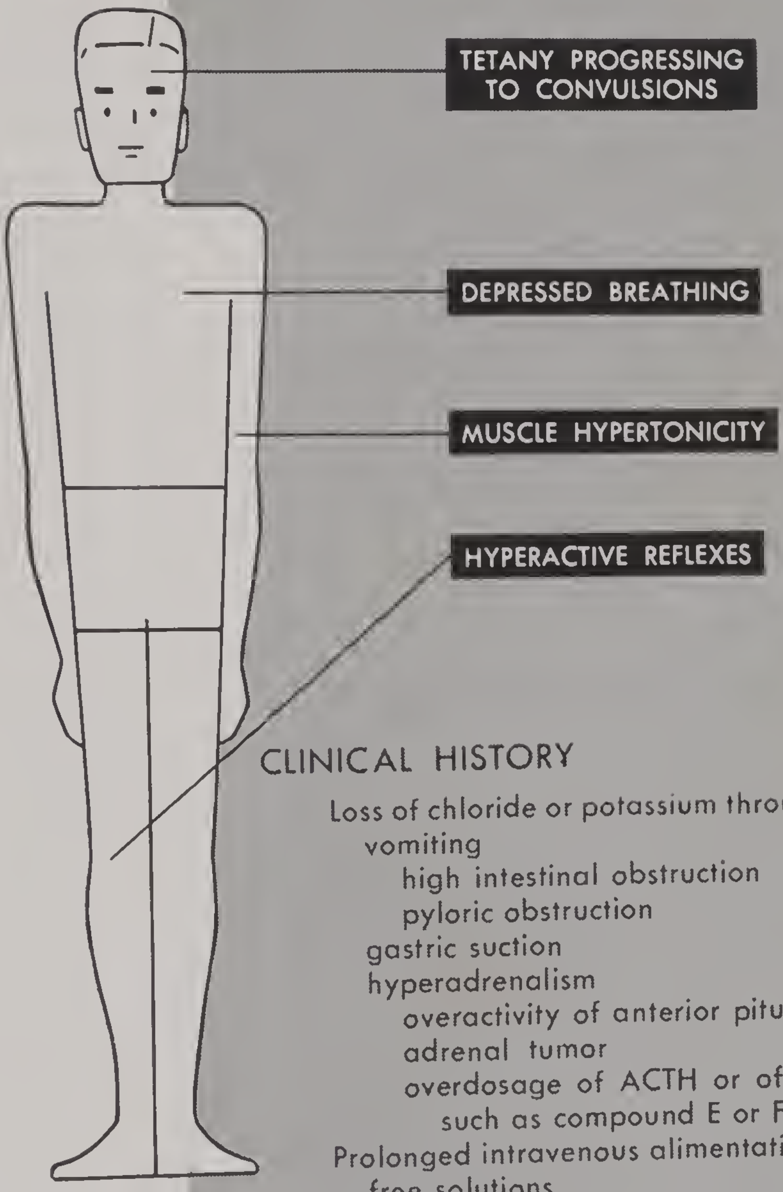
The increased alkalinity of the extracellular fluid decreases the ionization of calcium. Hence, tetany due to calcium deficit may be observed. In severe metabolic alkalosis, tetany may progress to convulsions. Muscle hypertonicity and hyperactive reflexes may be observed in base bicarbonate excess.

The urine becomes alkaline with pH above 7.0, determined conveniently by use of Nitrazine® paper. The urine may be acid in alkalosis when several conditions which are discussed in Chapter 15 are present. The pH of the plasma is found to be above 7.45. The plasma bicarbonate is above 29 mEq. per liter in adults and above 25 mEq. per liter in children. Note that the plasma bicarbonate reflects the amount of base bicarbonate.

In therapy of base bicarbonate excess (metabolic alkalosis)

CHANGES IN PROPERTIES OF EXTRACELLULAR FLUID

BASE BICARBONATE EXCESS (Metabolic Alkalosis)



CLINICAL HISTORY

- Loss of chloride or potassium through
  - vomiting
    - high intestinal obstruction
    - pyloric obstruction
  - gastric suction
  - hyperadrenalism
    - overactivity of anterior pituitary
    - adrenal tumor
    - overdosage of ACTH or of adrenal corticoids such as compound E or F
- Prolonged intravenous alimentation with potassium-free solutions
- Alkali therapy of peptic ulcer

LABORATORY FINDINGS

- pH of urine above 7.0
  - Nitrazine® paper gives pH reading by use of pH color comparison chart provided with papers
- pH of plasma above 7.45
- Plasma bicarbonate above 29 mEq./L. in adults and above 25 mEq./L. in children
- Plasma potassium may be below 4 mEq./L.

Figure 17. Clinical Picture of Base Bicarbonate Excess.

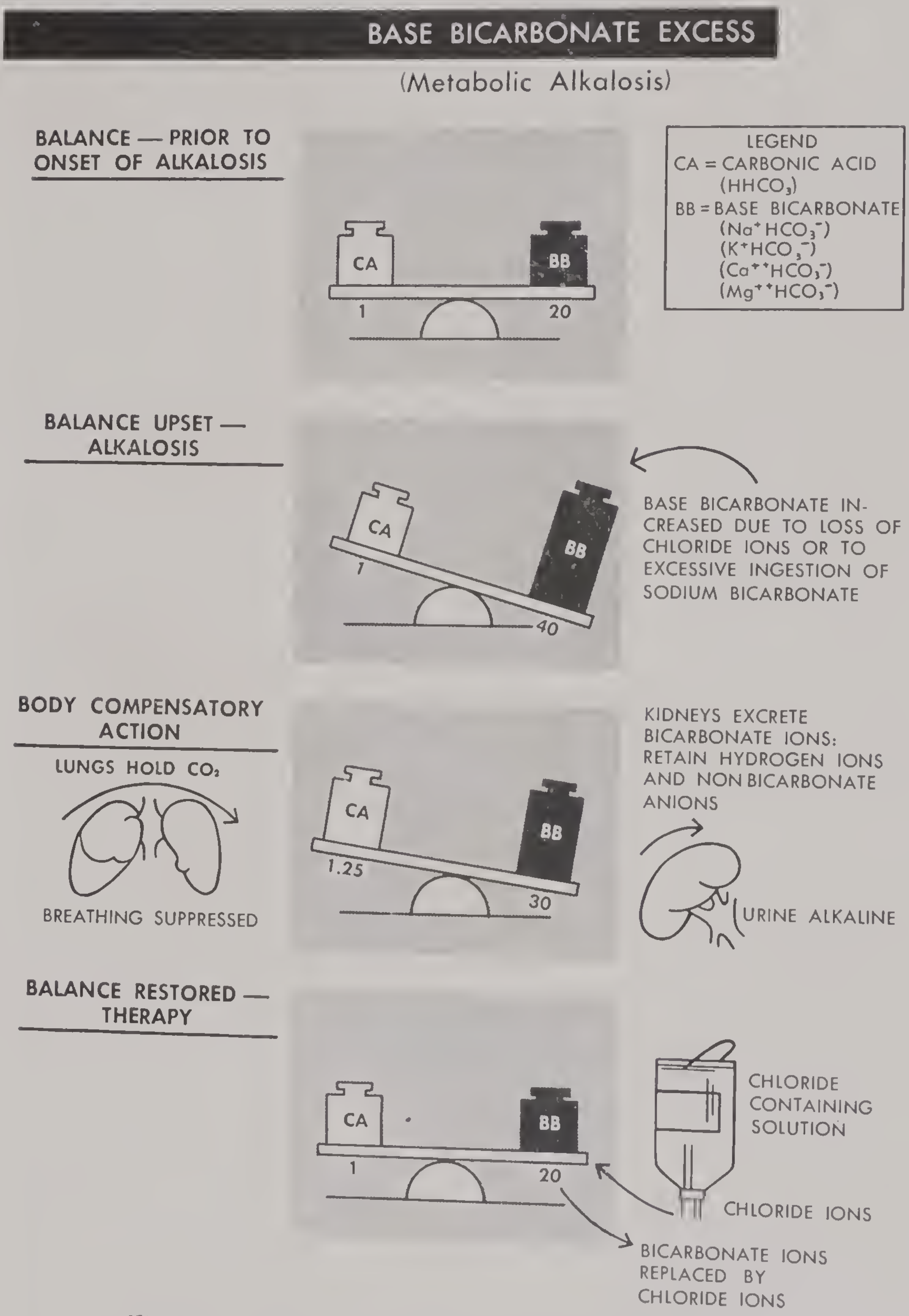

BASE BICARBONATE EXCESS
(Metabolic Alkalosis)
BALANCE — PRIOR TO ONSET OF ALKALOSIS
CA
BB
1
20
LEGEND
CA = CARBONIC ACID
($HHCO_3$)
BB = BASE BICARBONATE
($Na^+HCO_3^-$)
($K^+HCO_3^-$)
($Ca^{++}HCO_3^-$)
($Mg^{++}HCO_3^-$)
BALANCE UPSET — ALKALOSIS
CA
BB
1
40
BASE BICARBONATE INCREASED DUE TO LOSS OF CHLORIDE IONS OR TO EXCESSIVE INGESTION OF SODIUM BICARBONATE
BODY COMPENSATORY ACTION
LUNGS HOLD CO_2
BREATHING SUPPRESSED
CA
BB
1.25
30
KIDNEYS EXCRETE BICARBONATE IONS: RETAIN HYDROGEN IONS AND NON BICARBONATE ANIONS
URINE ALKALINE
BALANCE RESTORED — THERAPY
CA
BB
1
20
CHLORIDE CONTAINING SOLUTION
CHLORIDE IONS
BICARBONATE IONS REPLACED BY CHLORIDE IONS

Figure 18. Base Bicarbonate Excess Teeter-Totter.

a chloride-containing solution is given. The chloride ions of the solution replace bicarbonate ions and thus help relieve the primary base bicarbonate excess which is responsible for this imbalance. Since base bicarbonate excess is almost invariably accompanied by potassium deficit, potassium ions should be given. These may be incorporated in the solution of chloride or may be given in a separate solution later.

Carbonic Acid Deficit (Respiratory Alkalosis)

Prior to the onset of carbonic acid deficit, the teeter-totter is level (see Figures 19 and 20). Let us suppose that certain clinical conditions characterized by increased rate and depth of breathing occur. These may include oxygen lack, fever, high environmental temperature, encephalitis, the early stages of salicylate intoxication, voluntary overbreathing and hyperventilation due to hysteria or to anxiety. The latter is frequently seen in patients in respirators. Any of these conditions cause a blowing off of carbon dioxide through the lungs. This reduces the amount of carbonic acid in the extracellular fluid, and this carbonic acid deficit causes a tipping of the teeter-totter to the right. The pH rises to above 7.45. Alkalosis is present.

The carbonic acid deficit of respiratory alkalosis is *primary* since it is responsible for the disturbance of acid-base balance.

Body compensatory action begins. The lungs are unable to participate in this action since they are neutralized by the primary disease. The kidneys actively participate, however, and excrete bicarbonate ions while retaining hydrogen ions and nonbicarbonate anions. As a result the urine becomes alkaline. Because of the decreased ionization of calcium caused by the alkalinity of the extracellular fluid, tetany may occur. In severe respiratory alkalosis this may progress to convulsions.

The urine is alkaline, with a pH above 7.0, readily determined by use of Nitrazine® paper. The urine may be acid in this form of alkalosis as described in Chapter 15. The plasma bicarbonate is found to be below 25 mEq. per liter for adults and 20 mEq. per liter for children. The bicarbonate level reflects the amount of base bicarbonate on the teeter-totter.

In therapy of carbonic acid deficit, primary efforts should be directed at management of the pulmonary or systemic abnormality which caused the overbreathing initially. Parenteral fluid therapy may be of assistance. Chloride ions are given to replace bicarbonate ions and thus lighten the base bicarbonate side of the balance.

Note how, on the teeter-totter showing balance restored, the total amount of carbonic acid and base bicarbonate are each half of the amounts prior to the onset of alkalosis. The ratio, however, is 1 to 20 in spite of the differences in absolute amounts of carbonic acid and base bicarbonate, and the pH of the extracellular fluid is within normal limits.

The total amounts of carbonic acid and base bicarbonate shown prior to onset of the imbalance will not be restored until the disease that initially caused the imbalance is corrected. Before such restoration is achieved, the patient is said to be in "compensated respiratory alkalosis," in spite of the fact that the ratio of carbonic acid to base bicarbonate is 1 to 20 and the pH of the extracellular fluid lies between 7.35 and 7.45. In a sense the term is a misnomer.

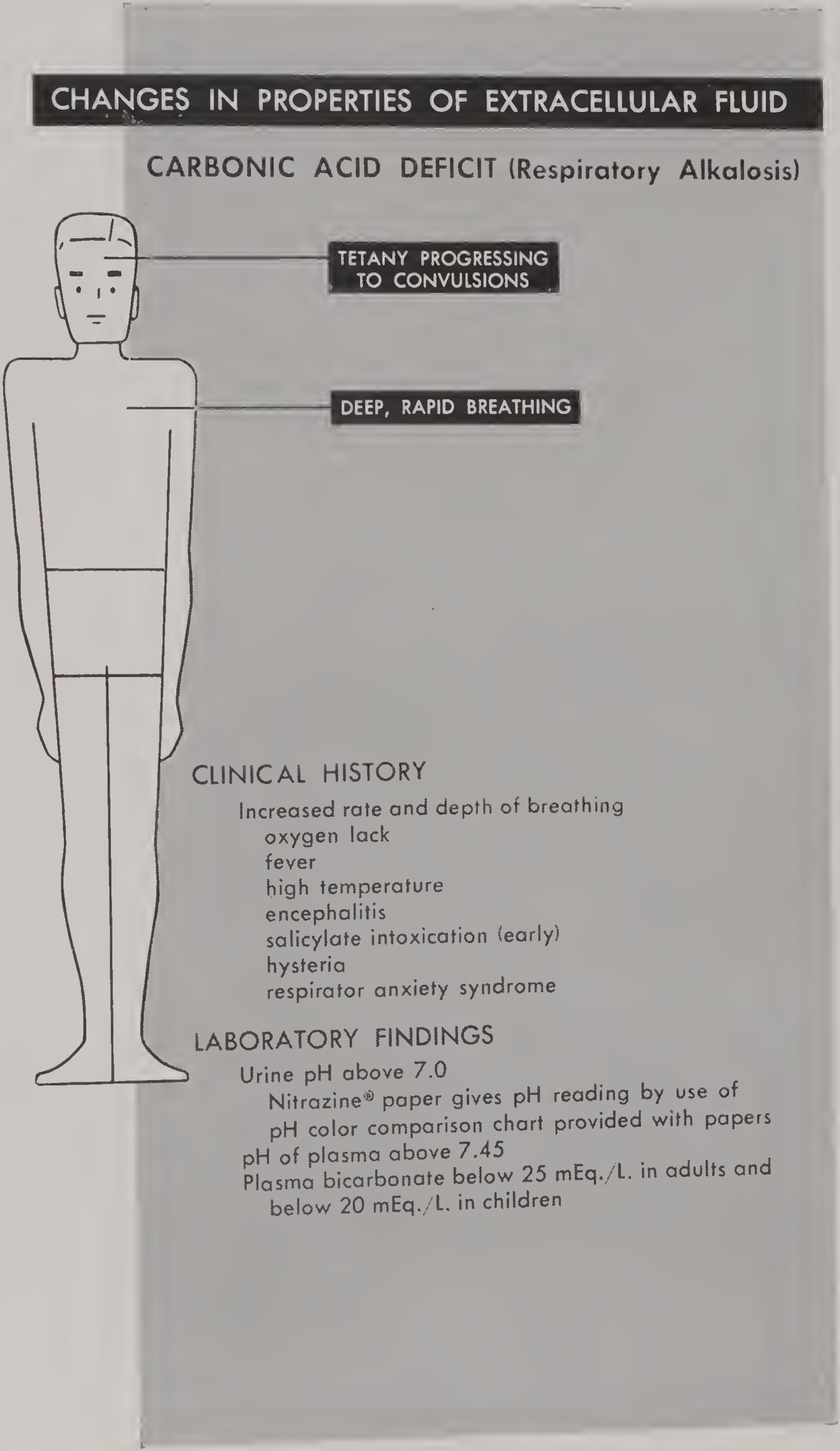
Carbonic Acid Excess (Respiratory Acidosis)

Before the onset of carbonic acid excess, we have 1 part of carbonic acid to 20 parts of base bicarbonate (see Figures 21 and 22). The teeter-totter is level. Several clinical conditions may cause a retention of carbon dioxide in the lungs. This causes an excess of carbonic acid, which is carbon dioxide plus water, in the extracellular fluid. Clinical conditions causing retention of carbon dioxide through impaired respiration include pneumonia, emphysema, paralysis of the muscles of respiration or of the bulbar portion of the medulla such as may occur in poliomyelitis, morphine poisoning, barbiturate poisoning, occlusion of the breathing passages, or the breathing of air rich in carbon dioxide. The teeter-totter is tipped to the left.

The pH of the extracellular fluid drops to below 7.35. Acidosis is present. The carbonic acid excess of respiratory acidosis is *primary* since it is responsible for the disturbance of acid-base balance.

Breathing is found to be suppressed. Other clinical findings include disorientation, weakness and, in severe imbalances, coma.

The lungs are unable to participate in body compensatory



CHANGES IN PROPERTIES OF EXTRACELLULAR FLUID

CARBONIC ACID DEFICIT (Respiratory Alkalosis)

CLINICAL HISTORY

Increased rate and depth of breathing
  oxygen lack
  fever
  high temperature
  encephalitis
  salicylate intoxication (early)
  hysteria
  respirator anxiety syndrome

LABORATORY FINDINGS

Urine pH above 7.0
  Nitrazine® paper gives pH reading by use of pH color comparison chart provided with papers
pH of plasma above 7.45
Plasma bicarbonate below 25 mEq./L. in adults and below 20 mEq./L. in children

Figure 19. Clinical Picture of Carbonic Acid Deficit.

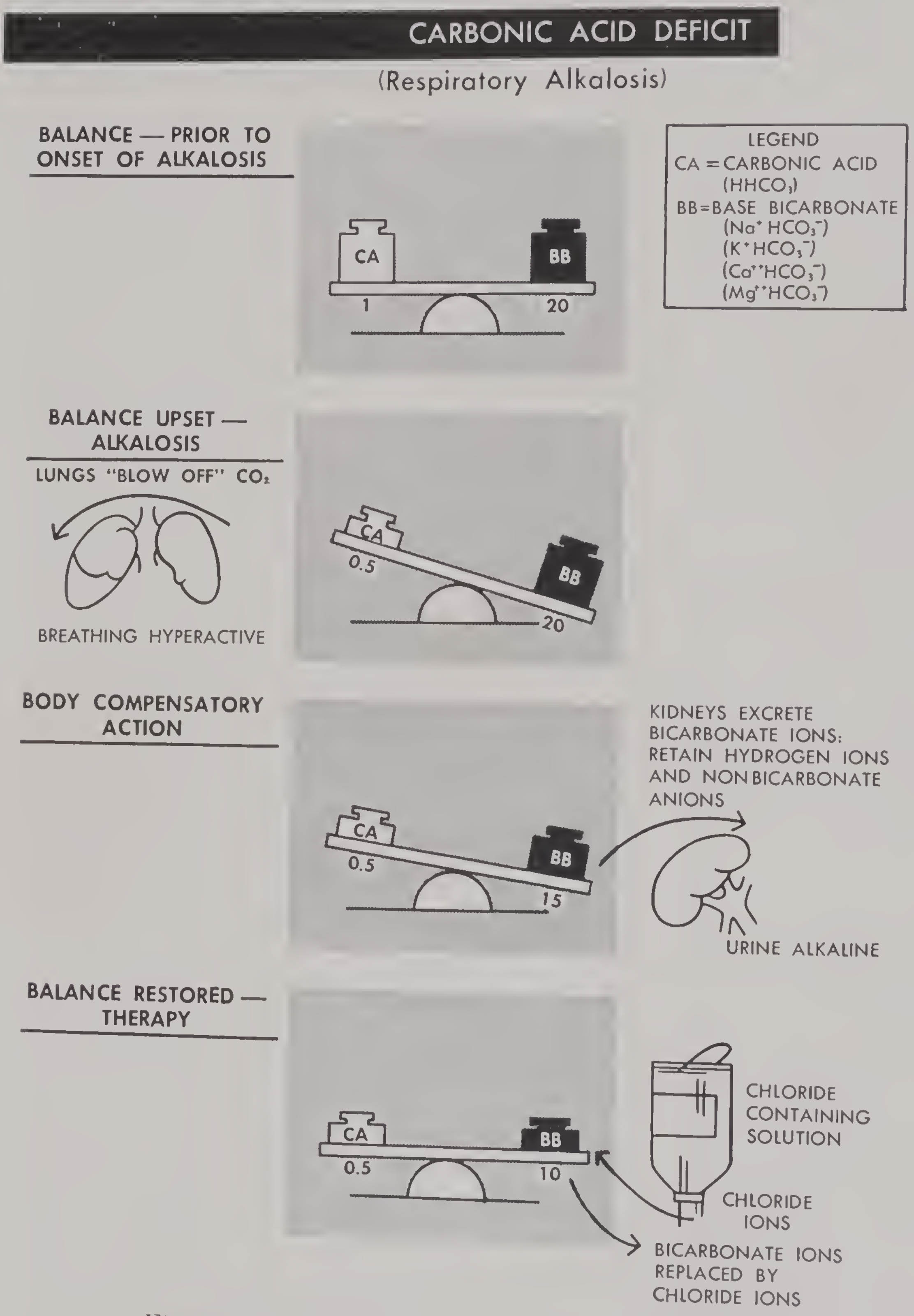
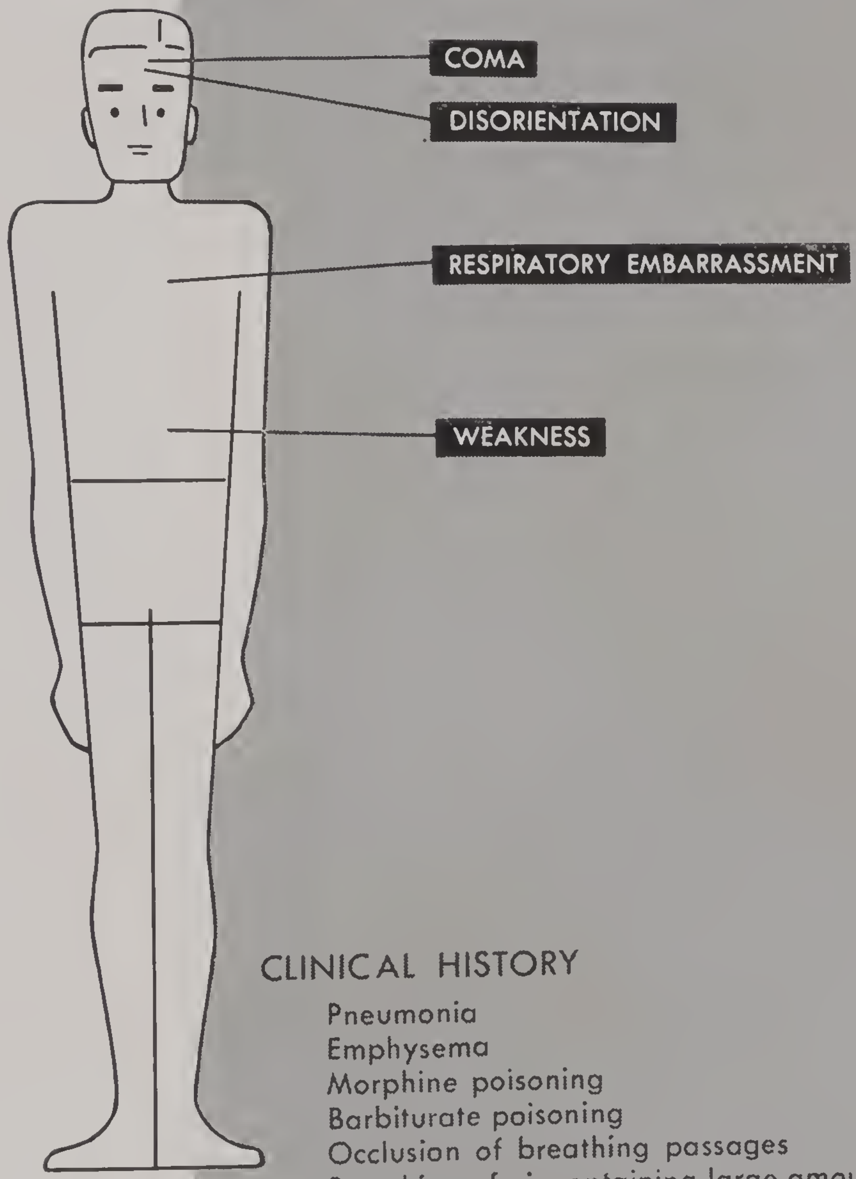

CARBONIC ACID DEFICIT
(Respiratory Alkalosis)
BALANCE — PRIOR TO ONSET OF ALKALOSIS
LEGEND
CA = CARBONIC ACID ($HHCO_3$)
BB = BASE BICARBONATE ($Na^+ HCO_3^-$) ($K^+ HCO_3^-$) ($Ca^{++} HCO_3^-$) ($Mg^{++} HCO_3^-$)
CA
BB
1
20
BALANCE UPSET — ALKALOSIS
LUNGS "BLOW OFF" CO_2
BREATHING HYPERACTIVE
CA
BB
0.5
20
BODY COMPENSATORY ACTION
KIDNEYS EXCRETE BICARBONATE IONS: RETAIN HYDROGEN IONS AND NONBICARBONATE ANIONS
CA
BB
0.5
15
URINE ALKALINE
BALANCE RESTORED — THERAPY
CHLORIDE CONTAINING SOLUTION
CHLORIDE IONS
CA
BB
0.5
10
BICARBONATE IONS REPLACED BY CHLORIDE IONS

Figure 20. Carbonic Acid Deficit Teeter-Totter.

CHANGES IN PROPERTIES OF EXTRACELLULAR FLUID

CARBONIC ACID EXCESS (Respiratory Acidosis)



CLINICAL HISTORY

- Pneumonia
- Emphysema
- Morphine poisoning
- Barbiturate poisoning
- Occlusion of breathing passages
- Breathing of air containing large amounts of carbon dioxide

LABORATORY FINDINGS

- Urine pH below 6.0
  - Nitrazine® paper gives pH reading by use of pH color comparison chart provided with papers
- pH of plasma below 7.35
- Bicarbonate above 29 mEq./L. in adults and above 25 mEq./L. in children

Figure 21. Clinical Picture of Carbonic Acid Excess.

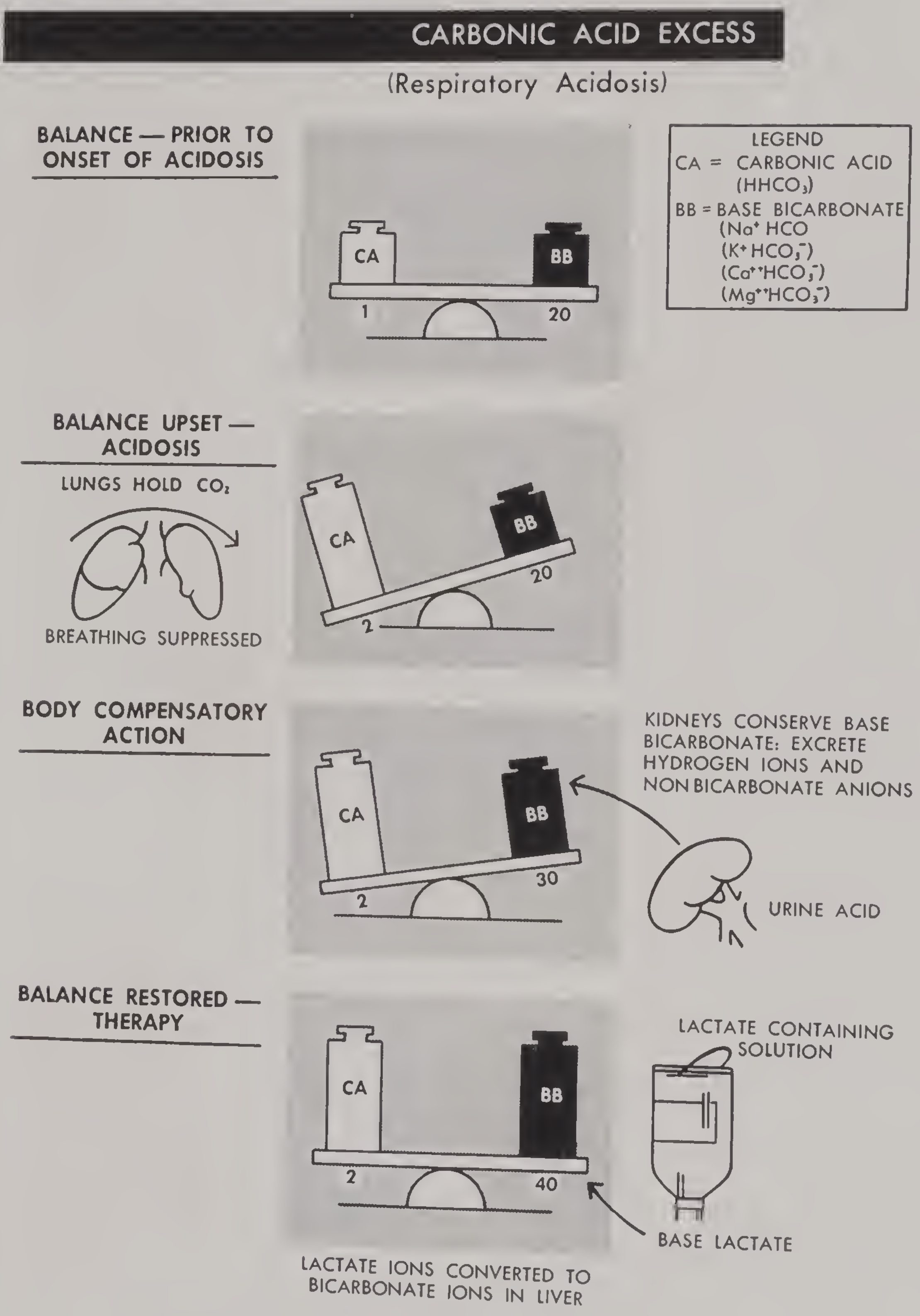


Figure 22. Carbonic Acid Excess Teeter-Totter.

action since they are not functioning normally. The kidneys attempt compensation by conserving base bicarbonate while excreting hydrogen ions and nonbicarbonate anions. As a result of this renal action, the urine becomes acid.

The urine pH is below 6.0, readily determined by use of Nitrazine® paper. The urine may be alkaline in this form of acidosis, as described in Chapter 15. The plasma pH is below 7.35. The bicarbonate content of the plasma is above 29 mEq. per liter for adults and above 25 mEq. per liter for children. Note how the bicarbonate value reflects the quantity of base bicarbonate on the teeter-totter.

Therapy should be primarily directed at relieving the pulmonary cause of the imbalance. If parenteral fluids are given, lactate ions are included in the solution. These are metabolized in the liver and are replaced by bicarbonate ions to cause a weighting of the base bicarbonate side of the teeter-totter.

Note how, on the teeter-totter showing balance restored, the total amounts of carbonic acid and base bicarbonate are each double the amounts that existed prior to the onset of acidosis. Since the ratio is 1 to 20, the pH of the extracellular fluid is within normal limits. The total amounts of carbonic acid and base bicarbonate shown prior to onset of the imbalance will not be restored until the pulmonary disease that initially caused the imbalance is corrected. Before such restoration is achieved, the patient is said to be in "compensated respiratory acidosis," in spite of the fact that the *ratio* of carbonic acid to base bicarbonate is 1 to 20 and the pH of the extracellular fluid lies between 7.35 and 7.45. This term, like the term "compensated respiratory alkalosis," is misleading.

SUMMARY

1) Each of the changes in the properties of extracellular fluid has its characteristic clinical history, physical findings and laboratory findings, including, in some instances, radiographic and electrocardiographic findings.

2) These changes, which are either deficits or excesses, involve volume, electrolyte concentration or electrolyte composition of extracellular fluid.

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Changes in Position of Extracellular Fluid

> *Disease at time creates experiments that physiology completely fails to duplicate, and the wise physiologist can obtain clues to the resolution of many problems by studying the sick.*
>
> Charles H. Mayo

PLASMA-TO-INTERSTITIAL FLUID SHIFT

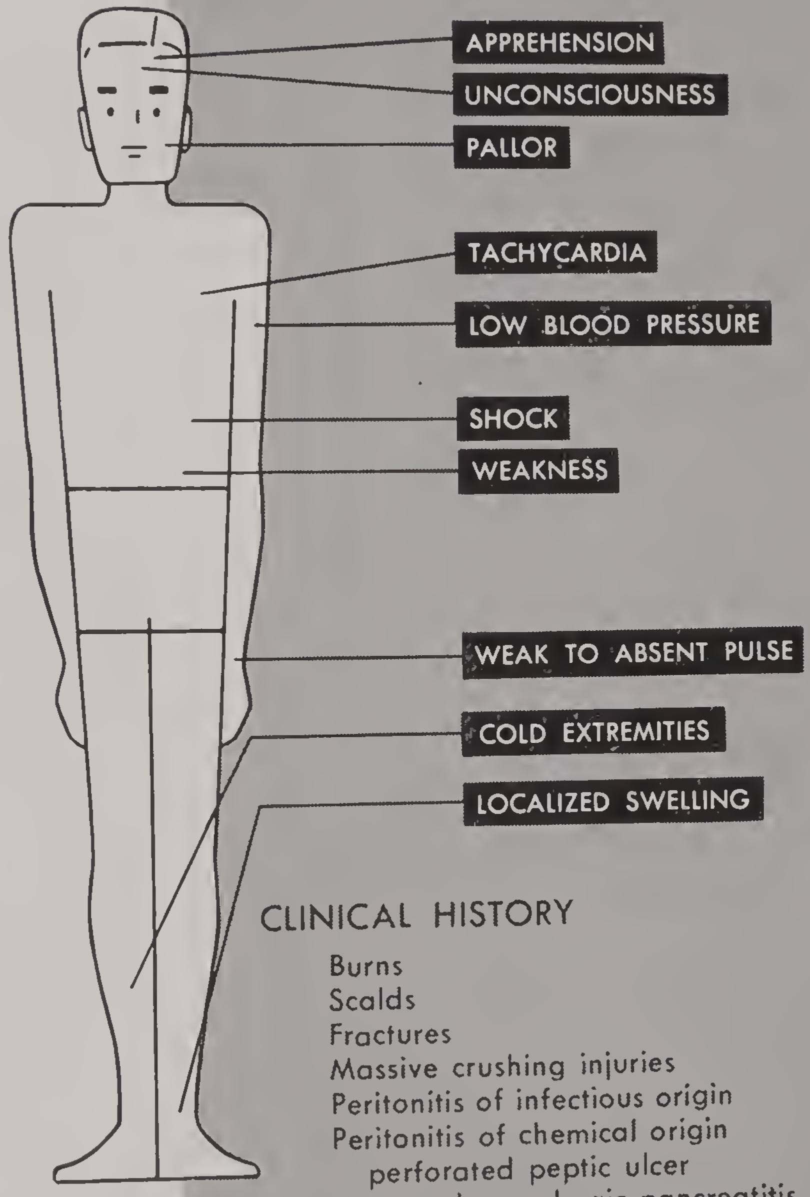
Normally, about one-fourth of the total extracellular fluid exists as plasma and about three-fourths as interstitial fluid. In certain clinical situations water and electrolytes shift from the plasma to the interstitial fluid. Clinical causes of such a shift include burns, scalds, fractures, massive crushing injuries, infectious peritonitis, peritonitis of chemical origin such as that caused by perforated peptic ulcer or acute hemorrhagic pancreatitis, pleuritis with effusion, dermatoses characterized by effusions of fluid into the skin, occlusion of large arteries, intestinal obstruction, and thromboses of large intraabdominal veins. The rapid accumulation of ascitic fluid which sometimes follows abdominal paracentesis represents such a shift.

The clinical findings are largely those of shock. They may include apprehension, pallor, weakness, low or absent blood pressure, tachycardia, weak or absent pulse, cold extremities and unconsciousness. If the shift has been localized, as in the swelling of a sprained ankle or of a fractured femur, the accumulation of interstitial fluid will be obvious upon physical examination.

The laboratory findings of a plasma-to-interstitial fluid shift include elevation of the hematocrit, the hemoglobin and the red blood cell count. The reason for these elevations lies in the fact that part of the water and electrolytes of the blood has been lost to the interstitial fluid leaving the formed elements in a smaller

CHANGES IN POSITION OF EXTRACELLULAR FLUID

PLASMA-TO-INTERSTITIAL FLUID SHIFT



CLINICAL HISTORY

- Burns
- Scalds
- Fractures
- Massive crushing injuries
- Peritonitis of infectious origin
- Peritonitis of chemical origin
  - perforated peptic ulcer
  - acute hemorrhagic pancreatitis
- Pleuritis with effusion
- Bullous or erythematous dermatitis
- Acute occlusion of major arteries
- Intestinal obstruction
- Thrombosis of portal or mesenteric veins
- Rapid accumulation of ascitic fluid after abdominal paracentesis

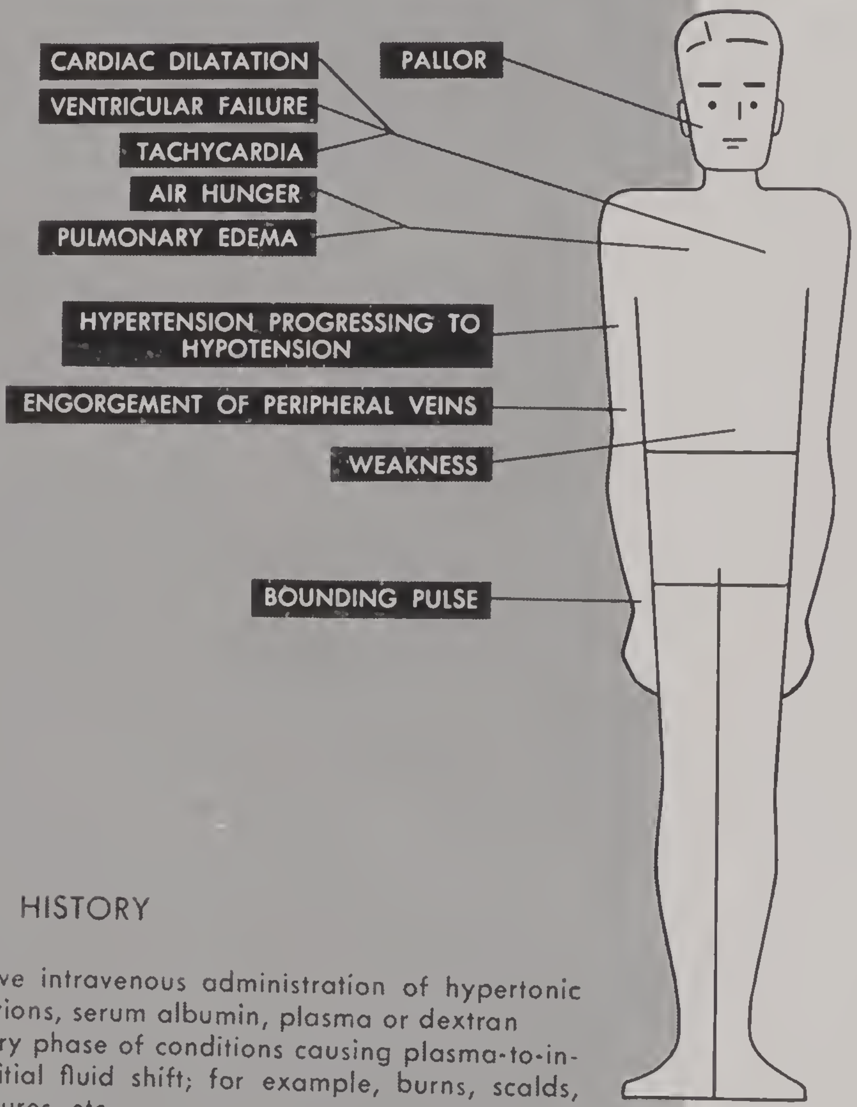
LABORATORY FINDINGS

- Hematocrit, hemoglobin and red blood cell count elevated

Figure 23. Clinical Picture of Plasma-to-Interstitial Fluid Shift.

CHANGES IN POSITION OF EXTRACELLULAR FLUID

INTERSTITIAL FLUID-TO-PLASMA SHIFT



CLINICAL HISTORY

Class I

Excessive intravenous administration of hypertonic solutions, serum albumin, plasma or dextran

Recovery phase of conditions causing plasma-to-interstitial fluid shift; for example, burns, scalds, fractures, etc.

Class II

Compensatory phase which may follow internal or external loss of whole blood

LABORATORY FINDINGS

Hematocrit, hemoglobin and red blood cell count decreased

Figure 24. Clinical Picture of Interstitial Fluid-to-Plasma Shift.

amount of plasma. Other findings include an increase in the level of plasma proteins above the normal of 8 Gm. per 100 ml. and an increase in the level of the plasma albumin above the normal 5 Gm. per 100 ml.

Therapy includes correcting the underlying cause of the shift and replacing the water and electrolytes of the plasma either by oral or parenteral administration of appropriate solutions. In addition, blood, dextran or plasma is given in order to restore the plasma volume and to draw back the shifted water and electrolytes from the interstitial fluid by increasing the osmotic pressure of the plasma. For the clinical picture of plasma-to-interstitial fluid shift see Figure 23.

INTERSTITIAL FLUID-TO-PLASMA SHIFT

Shifts of water and electrolytes from the interstitial fluid to the plasma are of two different classes. *The first class of shift* is characterized by a clinical history of excessive intravenous administration of hypertonic electrolyte solutions, serum albumin, plasma or dextran. This class of shift can also occur in the recovery phase of conditions which cause a plasma-to-interstitial fluid shift as discussed in the previous section. These conditions include burns, scalds, fractures and crushing injuries.

Upon physical examination of the patient suffering from the first class of shift, the physician may find hypertension which progresses to hypotension, pulmonary edema, bounding pulse, engorgement of peripheral veins, cardiac dilatation and, if the condition progresses, ventricular failure.

The second class of interstitial fluid-to-plasma shift represents a compensatory phase which may follow internal or external loss of whole blood. On physical examination, patients with this class of shift manifest pallor, weakness, air hunger and tachycardia.

In either class of interstitial fluid-to-plasma shift the hematocrit, hemoglobin and red blood cell count will be found to be decreased.

Therapy of both classes of this imbalance includes relief of excessive plasma volume by phlebotomy if the condition is critical. Both the hypotension and the cardiac dilatation should receive appropriate medical attention. Significant losses of whole blood should be corrected by administration of red blood cells or whole blood. For the clinical picture of interstitial fluid-to-plasma shift see Figure 24.

SUMMARY

There are two specific clinical fluid imbalances under the heading of changes in position of extracellular fluid, each with characteristic history, physical findings and laboratory findings.

13

Changes in the Nutritional Status of the Body

> *For that elephant ate all night,*
> *And that elephant ate all day;*
> *Do what he could to furnish him food,*
> *The cry was still more hay.*
>
> John Cheever Goodwin

The physician with a patient who must be nourished by the parenteral route for more than a few days can sympathize with the man who simply could not provide his elephant with enough hay. It is a difficult matter to get sufficient "hay," especially protein and sources of calories, into the patient when only the parenteral route is available. If maintenance of the nutrition of the healthy person is important, maintenance of the nutrition of the sick patient is doubly important. The further the physician can go toward meeting the nutritional needs of his patient the better, even though optimal nutrition appears impossible under the circumstances.

PROTEIN DEFICIT

Protein is an essential constituent of all living cells. The amino acids, of which it is composed, provide the building units for growth. Without protein there can be no synthesis of new tissue and no repair of damaged or destroyed tissue. Because of its unique position in the nutritional arch, we have termed it the "keystone nutrient."

The clinical history of the patient with a protein deficit will reveal one or more conditions causing decreased intake, increased loss or impaired utilization of protein. Such conditions include severe, repeated or chronic hemorrhage, chronic or repeated infections, starvation, extensive burns, fractures, draining wounds, decubitus ulcers, repeated surgical procedures, lipoid nephrosis, nephrotic syndrome or hyperthyroidism. Malignancy or prolonged gastrointestinal disease may also be responsible for a severe protein deficit.

Clinical findings include loss of weight, loss of muscle mass and tone, and loss of tissue turgor. The patient may be pale and edematous. He may suffer from malaise and chronic fatigue. Anorexia and cyclic vomiting may be present. Impaired wound healing and decreased resistance to infection frequently accompany body protein deficit. Furthermore, protein deficit may inhibit the development of callus following fractures and may increase susceptibility to hemorrhagic shock.

While the diagnosis of a body protein deficit is chiefly based on the clinical history and results of physical examination, a plasma albumin level below 4 Gm. per 100 ml. should arouse strong suspicion that this imbalance exists. Hematocrit, hemoglobin and red blood cell count are usually below normal, not only in severe protein deficits, but in moderate protein deficits as well.

The goal of therapy is to supply protein plus sufficient calories to insure that amino acids will not be used for energy purposes. The enteral route is preferred, whether for oral or tube feedings. It is difficult to replace depleted protein stores by the parenteral route alone, although administration of solutions containing amino acids and carbohydrates can prevent further development of protein deficit in most patients restricted to parenteral feedings. For the clinical picture of protein deficit see Figure 25.

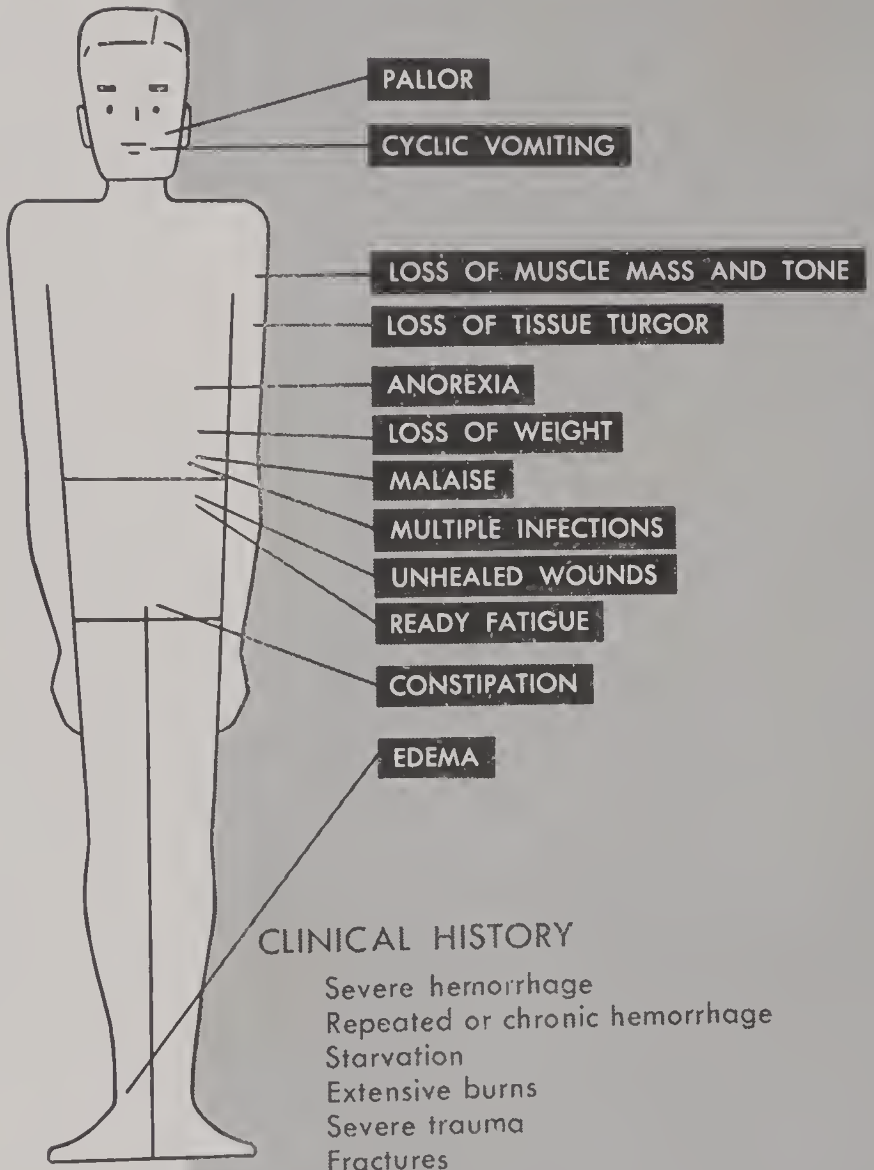
CALORIC DEFICIT

The clinical causes of caloric deficit include decreased food intake, gastrointestinal disease, hyperthyroidism, chronic infectious disease, severe burns and severe trauma.

When the patient's caloric intake is inadequate, protein is used to meet energy needs. Either parenterally administered protein or protein and fat of the body tissues may be so utilized, if calories are not provided in the form of carbohydrate, fat or alcohol. For this reason, the clinical findings of caloric deficit may be partly caused by protein deficit and partly by caloric deficit. These find-

CHANGES IN NUTRITIONAL STATUS OF THE BODY

PROTEIN DEFICIT



CLINICAL HISTORY

Severe hemorrhage
Repeated or chronic hemorrhage
Starvation
Extensive burns
Severe trauma
Fractures
Wound drainage
Decubitus ulcers
Repeated surgery

LABORATORY FINDINGS

Plasma albumin level below 4.0 Gm./100 ml.
Hematocrit, hemoglobin and red blood cell count below normal

Figure 25. Clinical Picture of Protein Deficit.

ings include loss of weight, loss of muscle mass and tone, impaired wound healing, shortness of breath, weakness, mental depression and apprehension.

Laboratory findings may include acetonuria, if the patient is utilizing his own tissues. Any serious caloric deficit which has been present for more than a limited period will result in a protein deficit due to the metabolism of body tissues for energy purposes. Sources of calories are ideally supplied by the diet. Excellent tube feeding mixtures are available. Calories can also be supplied in parenteral solutions containing suitable mixtures of dextrose, fructose and alcohol. For the clinical picture of caloric deficit see Figure 26.

VITAMIN DEFICIT

The clinical causes of vitamin deficits include decreased intake of food sources of vitamins, gastrointestinal disease, chronic infectious disease, severe burns and severe trauma.

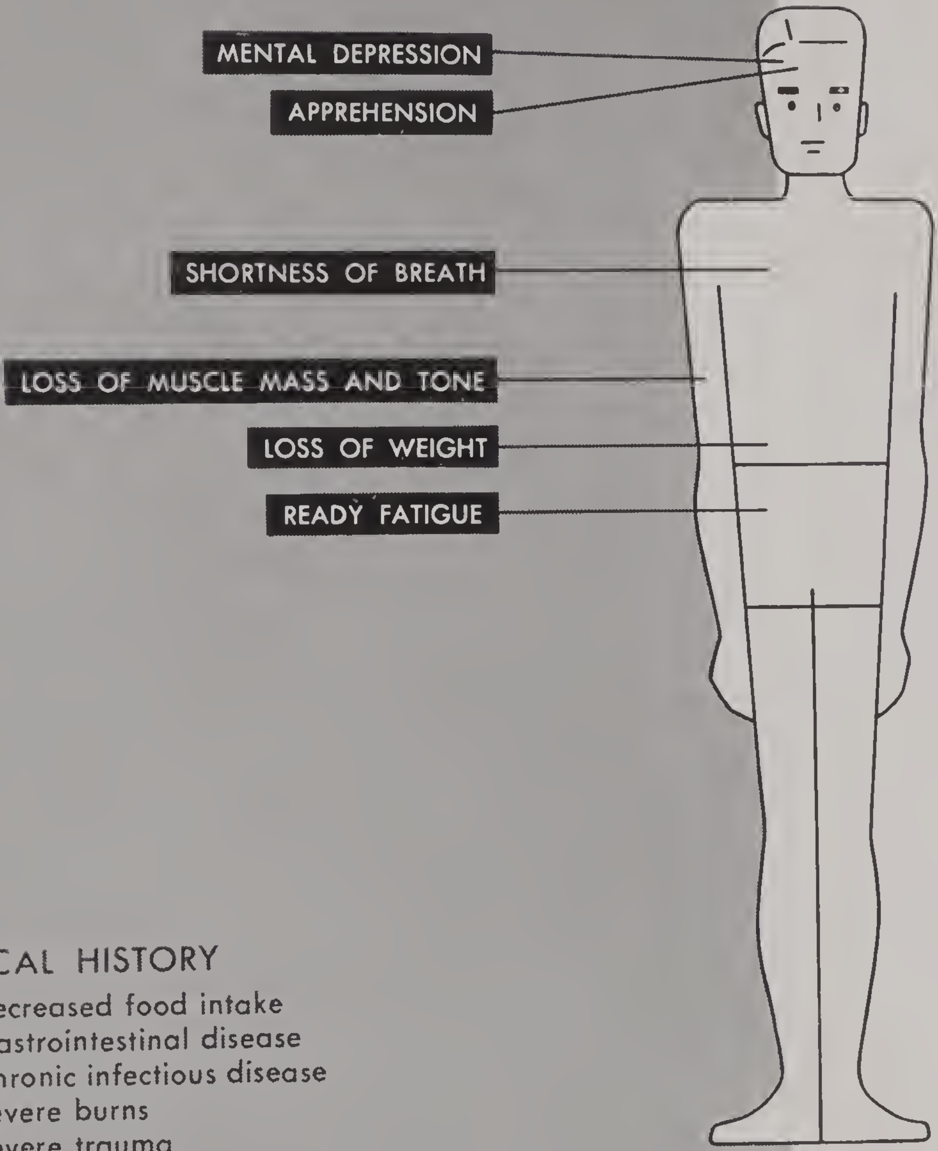
Physical findings depend upon the specific vitamin deficiency present. Vitamin A deficit may be manifested by night blindness. Manifestations of deficiency of B vitamins include cheilosis or cracks at the mouth corners, beefy-red tongue, diarrhea, anemia, dermatitis of exposed areas and anorexia. Vitamin C deficiency may be indicated by bleeding gums or perifollicular hemorrhages; subperiosteal hemorrhages are found in advanced stages of the deficiency in infants. Impaired wound healing has been attributed to deficiency of vitamin C. Bowed legs, Harrison's groove and prominent frontal bosses are characteristic of vitamin D deficiency rickets. It is important to recall that vitamin deficiencies are often multiple.

Laboratory findings indicative of vitamin deficit include characteristic epiphyseal changes, as seen in roentgenograms, both in vitamin C and vitamin D deficiency.

When a vitamin deficit is present, it is usually readily managed by administration of a concentrated multivitamin preparation, either parenterally or by mouth. Some parenteral solutions have vitamins incorporated in them. For the clinical picture of vitamin deficit see Figure 27.

CHANGES IN NUTRITIONAL STATUS OF THE BODY

CALORIC DEFICIT



CLINICAL HISTORY

Decreased food intake
Gastrointestinal disease
Chronic infectious disease
Severe burns
Severe trauma

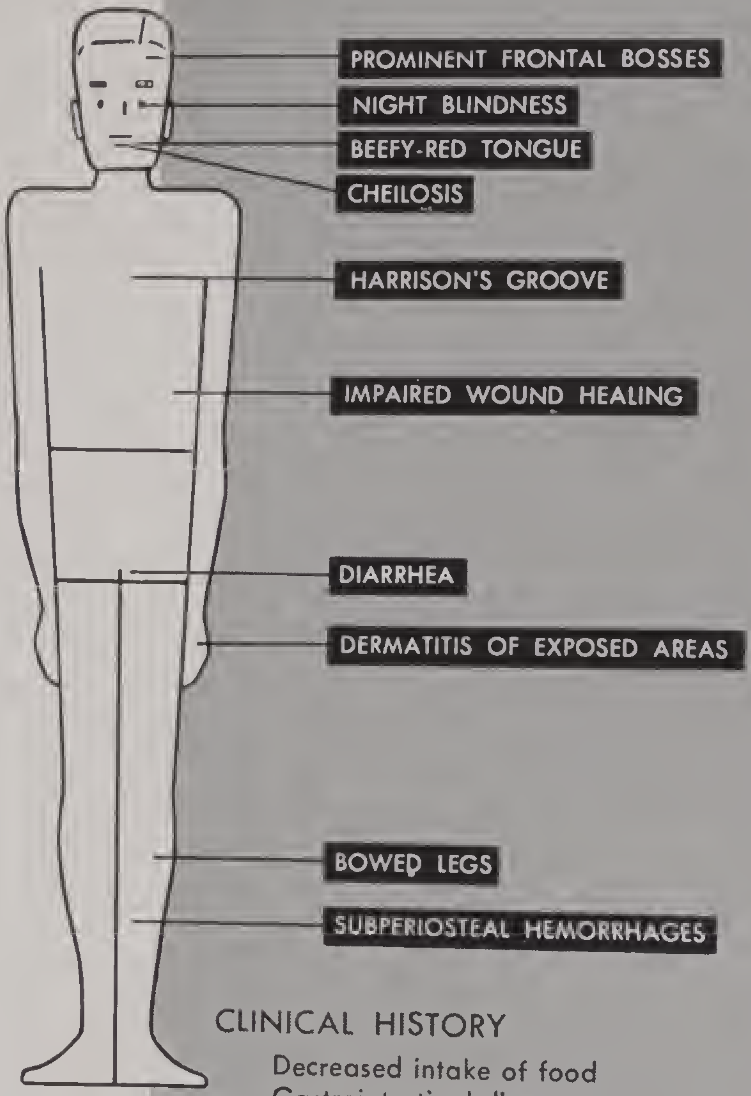
LABORATORY FINDINGS

Acetonuria
Findings of protein deficit, which ultimately results from caloric deficiency

Figure 26. Clinical Picture of Caloric Deficit.

CHANGES IN NUTRITIONAL STATUS OF THE BODY

VITAMIN DEFICIT



CLINICAL HISTORY

Decreased intake of food
Gastrointestinal disease
Chronic infectious disease
Severe burns
Severe trauma

LABORATORY FINDINGS

X-ray reveals typical findings of vitamin C deficiency (scurvy) and vitamin D deficiency (rickets)

Figure 27. Clinical Picture of Vitamin Deficit.

SUMMARY

1) Each of the changes in the nutritional status of the body has its own characteristic clinical history, physical findings and laboratory findings.

2) These imbalances are all too frequently forgotten, since they are slow in developing and do not, of themselves, produce acute emergencies.

3) Frequently, the manner in which nutritional status changes are managed decides the issue between life and death for the patient.

14

Systematic Diagnosis

> *The horror of that moment, the King went on, I shall never, never forget! You will though, the Queen said, if you don't make a memorandum of it.*
>
> Lewis Carroll

Chassin has well pointed out: "Intelligent interpretation of the patient's history, his clinical manifestations and his intake-output chart is far more essential to proper electrolyte therapy than is the performance of a multitude of chemical determinations on the patient's blood or plasma." Nevertheless, all available evidence should be considered in arriving at a diagnosis and before proceeding with treatment. We shall discuss in this chapter the systematic approach to the clinical history, the physical findings and the laboratory findings.

CLINICAL HISTORY

The history is of cardinal importance. From his knowledge of the effect which diseases of major organ systems exert on the properties of extracellular fluid, on the position of extracellular fluid and on the nutritional status of the body, the physician can often determine from the history alone the approximate body fluid status of the individual patient.

The *skin,* for example, is an organ from which water is constantly lost as insensible perspiration. *Febrile illnesses* may increase this loss. If not replaced, water lost in this manner causes a fluid volume deficit and may play a major role in the development of electrolyte concentration excess. *Sweating,* or sensible perspiration, is a route of water, sodium and chloride loss. Replacement of these losses by water alone causes an electrolyte concentration deficit.

Disease of the *central nervous system* may indirectly result in fluid imbalances. For example, the *unconscious* or *disoriented patient* may develop a fluid volume deficit because of inadequate fluid intake. The patient with *bulbar paralysis* may develop a fluid volume deficit because of his inability to swallow. *Increased intracranial pressure* causes vomiting with its attendant loss of water, chloride and potassium. *Encephalitis* accompanied by rapid breathing may result in carbonic acid deficit (respiratory alkalosis).

Cardiovascular dysfunction is often associated with body fluid imbalances. The fluid volume excess of *congestive heart failure* is a familiar example. Overtreatment of congestive heart failure by a low sodium diet and mercurial diuretics leads to electrolyte concentration deficit.

Water as well as carbon dioxide is lost through the lungs. *Febrile illnesses, inflammatory diseases* of the lungs, *salicylate intoxication* and diseases associated with a base bicarbonate deficit (metabolic acidosis) are often accompanied by an increased rate and depth of respiration. Increased water loss accompanies such hyperventilation; fluid volume deficit and electrolyte concentration excess may result. Excessive loss of carbonic acid, leading to carbonic acid deficit (respiratory alkalosis), accompanies the hyperventilation of *hysteria, salicylate intoxication* and *encephalitis.* Carbonic acid excess (respiratory acidosis) accompanies hypoventilation resulting from *drug depression* of the respiratory center or intrinsic disease of the lungs such as *emphysema.*

The *gastrointestinal tract* is often the source of water and electrolyte loss. The loss of water in excess of electrolytes which accompanies *watery diarrhea* often results in fluid volume deficit and electrolyte concentration excess as well as in potassium deficit. *Vomiting* and *gastric suction* with loss of water, chloride and potassium may result in fluid volume deficit, base bicarbonate excess (metabolic alkalosis) and potassium deficit. The *steatorrhea* of sprue or celiac disease may lead to a calcium deficit. Acute *caloric deficits* accompanying poor food intake or absorption are associated with development of base bicarbonate deficit (metabolic acidosis) because of attendant *ketosis.*

Disease of the *kidney* with consequent loss of its ability to selectively retain or reject water, electrolytes and metabolic waste products may be accompanied by a host of fluid imbalances. For example, base bicarbonate deficit (metabolic acidosis) and potassium excess accompany *chronic glomerular nephritis.* Protein deficit and fluid volume excess are characteristic of *childhood nephrosis.*

Dysfunction of various *glands of internal secretion* often results in fluid imbalances. For example, uncontrolled *diabetes mellitus* may cause fluid volume deficit, base bicarbonate deficit (metabolic acidosis) and caloric deficit. Potassium excess accompanies *adrenal insufficiency* and potassium deficit is seen with *hyperadrenalism.*

Intake of water and electrolytes is also important. Fluid volume deficit accompanies *acute cessation of fluid intake.* Base bicarbonate deficit (metabolic acidosis) may accompany ingestion of ammonium chloride by the patient with *heart disease.* Excessive ingestion of sodium bicarbonate by the patient with a *peptic ulcer* may lead to base bicarbonate excess (metabolic alkalosis). *Over-enthusiastic use of parenteral fluids* may lead to fluid volume excess.

Knowledge, not only of the quantity of fluids gained or lost but of their composition, is of significance in arriving at a correct diagnosis. Usually the quantity of such losses will have to be estimated. The more accurate the estimation the better from the standpoint of accurate diagnosis.

Perusal of the visual summaries of the specific fluid imbalances presented by Figures 7 to 27 will reveal many more situations in which the presence of a certain disease, as discovered in the clinical history, suggests the presence of one or more specific fluid imbalances.

PHYSICAL EXAMINATION

Equal in importance to the clinical history is the physical examination. A systematic approach to this examination is as vital in the diagnosis of fluid imbalance as it is in the diagnosis of other types of disorders.

Evaluation of the patient's *sensorium* is a logical first step. Lassitude or stupor may suggest a fluid volume deficit. Apprehension and headache may indicate an electrolyte concentration deficit. Weakness and disorientation may give the clue to a base bicarbonate deficit (metabolic acidosis).

The presence or absence of *thirst* is important. While thirst frequently indicates that the body needs fluids, one cannot assume that when thirst is absent there is no need for fluids. As Holmes and Montgomery pointed out in their classic article "Thirst as a Symptom," thirst may be important to diagnosis or therapy not only in fluid volume deficits but in cardiac edema, polyurias of endocrine origin, hemorrhage, diseases altering salivary secretion, and emotional disturbances.

The *skin* deserves careful appraisal. Pitting edema may be found in fluid volume excess or in protein deficit. The skin is flushed in electrolyte concentration excess. The presence of a dermatitis of exposed areas may lead to a diagnosis of a vitamin deficit of niacin. Fingerprinting of the skin over the sternum suggests a cellular fluid volume excess which may accompany a deficit in total electrolyte concentration of the extracellular fluid.

Body weight gains and losses are important. A loss up to 10% of the weight of an infant or a small child suggests a moderate extracellular or body fluid volume deficit. A weight loss above 10% indicates a severe fluid volume deficit. A weight loss up to 5% in a larger child or adult suggests a moderate fluid deficit and a weight loss above 5% a severe body fluid volume deficit.

The *mucous membranes* offer helpful clues. When the tongue is shrunken like a "lemon in the sun," extracellular fluid volume deficit is suggested. Excessive lacrimation and excessive salivation may give the clue to an electrolyte concentration deficit. When the mucous membranes are dry and sticky, electrolyte concentration excess may be present.

That faithful pump, the *heart*, should have its day in court. Tachycardia is not specific but may give the lead to a plasma-to-interstitial fluid shift of water and electrolytes. Cardiac dilatation may be found in a rapid interstitial fluid-to-plasma shift.

Closely related to the cardiac findings are those of the *blood vessels.* Engorgement of the peripheral veins immediately suggests an excess of extracellular fluid volume. The hypotension of a plasma-to-interstitial fluid shift with shock is frequently observed in accident wards. Hypertension progressing to hypotension may be seen in the opposite shift, from interstitial fluid-to-plasma.

Examination of the *lungs* may reveal the moist rales of fluid volume excess. Rapid breathing may be present in either carbonic acid deficit (respiratory alkalosis) or base bicarbonate deficit (metabolic acidosis). Slow, shallow respirations may indicate base bicarbonate excess (metabolic alkalosis). Laryngeal stridor calls urgent attention to the possibility of a calcium deficit.

The *stomach and intestines* provide many clues. Vomiting is frequently responsible for base bicarbonate excess (metabolic alkalosis) or potassium deficit. Intestinal colic should warn of potassium excess, and silent intestinal ileus and gaseous distention of the intestines are typical of potassium deficit.

Physical examination should not neglect those organs of homeostasis par excellence, the *kidneys.* Pain or tenderness in the area overlying them may suggest the renal concretions of calcium excess or infection. The latter may lead to potassium excess if renal function is impaired. Oliguria may indicate a fluid volume deficit or an electrolyte concentration excess. It is also present, more ominously, in potassium excess.

The *muscles* are putty-like in fluid volume deficit. They resemble half-filled water bottles in severe potassium deficit. Loss of muscle mass and tone suggests protein deficit. Tetany or cramps of the muscles may be present in calcium deficit and hypotonicity of the muscles occurs in calcium excess.

The *bones,* dynamic and living structures that they are, make their contribution to diagnosis of specific fluid imbalances. Deep bony pain caused by osteoporosis or bony cavitation may be present in calcium excess. Tender swelling over the bones of infants may suggest the subperiosteal hemorrhage of severe vitamin C deficit.

In examination of the *nervous system* the hyperactive reflexes of base bicarbonate excess (metabolic alkalosis) and of calcium deficit should be borne in mind. Tingling of the fingers and circumoral area may give the lead to a calcium deficit.

Finally, the *body temperature* should be taken. It is often low in a fluid volume deficit. An elevated temperature may give the clue to an excess in electrolyte concentration or to a carbonic acid deficit (respiratory alkalosis).

Very young infants frequently do not react to disturbances of fluid balance with the signs and symptoms that are typical of older individuals; for example, the Kussmaul breathing of metabolic acidosis is an inconstant finding in young infants suffering from this imbalance. For this reason, greater reliance must often be placed on laboratory findings, both in diagnosis and therapy of immature infants with fluid balance problems, than with older infants, children and adults.

The above examples show how the findings on physical examination may give important clues to the presence of specific fluid imbalances. A fairly complete list is given in the visual summaries of specific fluid imbalances (see Figures 7 to 27).

LABORATORY FINDINGS

While laboratory findings in general are confirmatory rather than diagnostic, they are helpful, particularly when they can be obtained promptly. The principal laboratory values and their usual trends in specific fluid imbalances are listed in the chart showing laboratory values useful in diagnosis (see Table 1). The designation "I" stands for "increased over the normal" and the designation "D" for "decreased." Blank spaces indicate that the laboratory value in question is not ordinarily useful in the imbalance in question. As will be indicated in the next chapter, laboratory values should always be taken with a generous grain of sodium chloride. A single value is seldom of help. Serial determinations are essential.

APPROACH TO DIAGNOSIS

It is essential to proceed in a systematic manner in the study of the patient with imbalanced body fluids if an accurate, complete diagnosis is to be obtained. First, a careful clinical history should be taken, jotting down facts suggestive of specific imbalances. The past intake and output of fluids should be reviewed, with particu-

LABORATORY VALUES USEFUL IN DIAGNOSIS

Specific Imbalances

	Extracellular Fluid Property Changes												*Extracellular Fluid Position Changes*		*Nutritional Status Changes*		
	Volume		*Concentration*		*Composition*								*Shift*				
					Potassium		*Calcium*		*Base Bicarbonate*		*Carbonic Acid*						
	Deficit	*Excess*	*Deficit*	*Excess*	*Deficit*	*Excess*	*Deficit*	*Excess*	*Deficit*	*Excess*	*Deficit*	*Excess*	*Plasma to Interstitial Fluid*	*Interstitial Fluid to Plasma*	*Protein Deficit*	*Caloric Deficit*	*Vitamin Deficit*
RBC:4.3—6.0 million/cu.mm.	I	D											I	D	D		
Hb:13—16 Gm./100 ml.	I	D											I	D	D		
Hematocrit:37—54%	I	D											I	D	D		
Plasma Na⁺:137—147 mEq./L.			D	I													
Plasma K⁺:4.0—5.6 mEq./L.					D	I											
Plasma Ca⁺⁺:4.5—5.8 mEq./L.							D	I									
Plasma Cl⁻:98—106 mEq./L.			D	I						D							
Plasma phosphorus (inorganic): 1.7—2.6 mEq./L.							I	D									
Plasma protein: 6—8 Gm./100 ml. (16 mEq./L.)	I	D		I									I	D	D	D	
Plasma albumin: 4—5 Gm./100 ml.	I	D		I									I	D	D	D	
Plasma HCO_3^-: Adults: 25—29 mEq./L. Children: 20—25 mEq./L.									D	I	D	I					
Plasma $Cl^- + HCO_3^-$: 123—135 mEq./L.			D	I													
Plasma pH:7.35—7.45									D	I	I	D					
Urine pH:6—7									D	I	I	D					
Urine specific gravity: 1.010—1.030	I	D	D	I													

Only the principal laboratory values and their usual trends are listed.

Legend: I = increase in this disturbance
D = decrease in this disturbance

lar emphasis on the previous one or two days. Answers to these questions should be obtained:

- Has the patient been eating and drinking normally?
- Have therapeutic fluids been given, by tube, rectum or parenterally?
- Have abnormal losses of body fluids occurred as abnormal sweating, vomitus, gastric or intestinal suction, enterostomy drainage, liquid stools, wound or burn exudate?

Usually little more than a rough estimate of the intake-output record of the patient can be obtained through the history; even such an estimate can be of great value.

In carrying out the physical examination, the physician should list findings that may indicate a specific fluid imbalance. Laboratory findings should be studied, using table 1 for evaluation of their possible significance.

Table 2 is a simple, though adequate, *daily* (24-hour) *fluid balance sheet.* It can be employed for recording the initial intake-output estimate and the initial laboratory determinations, as well as for subsequent daily intake-output figures and laboratory determinations.

Now, with pertinent data available from the clinical history, physical examination and laboratory values, the physician has a solid basis for a systematic diagnosis, which should include both the imbalances present and their approximate severity. For example, the diagnosis on a given patient might be written as follows:

Changes in Properties of Extracellular Fluid

- Moderate volume deficit
- Slight electrolyte concentration excess
- Slight base bicarbonate deficit

Changes in Position of Extracellular Fluid

- None

Changes in Nutritional Status of Patient

- Moderate protein deficit

Close familiarity with the information presented by Figures 7 to 27 will help greatly in diagnosis.

TABLE 2

Daily Fluid Balance Sheet

Admission Diagnosis________________ Name or Number________________

Sex______ Age______ Height__________ Weight__________ Surface Area________________

	24-Hour Intake—output Record	First 12 Hours	Second 12 Hours	Total 24 Hours	Laboratory Findings	
G	Oral liquids					
A	Tube feedings				RBC: million/cu. mm.	
I	Rectal feedings				Hb: Gm./100 ml.	
N	Parenteral feedings				Hematocrit: Per cent	
S					Plasma Na$^+$: mEq./L.	
					Plasma K$^+$: mEq./L.	
L	Urine				Plasma Ca^{++}: mEq./L.	
O	Insensible loss*				Plasma Phosphorus: mEq./L.	
S	Vomitus				Plasma Protein: Gm./100 ml.	
S	Gastric suction				Plasma Albumin: Gm./100 ml.	
E	Intestinal suction				Plasma HCO$_3^-$: mEq./L.	
S	Enterostomy drainage				Plasma Cl$^-$+HCO$_3^-$: mEq./L.	
	Diarrheal stools				Plasma pH	
	Abnormal sweating**				Urine pH	
	Wound exudate				Urine Specific Gravity	
	Burn exudate					
	Fistula drainage					
	Other					

Notes: Record quantities as estimated or measured in milliliters. Such sources of gains as water from solid foods and chemical oxidation, and such losses as water in normal stools and normal perspiration are not included because of relative unimportance and difficulty of measurement.

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*Insensible loss (insensible perspiration through skin and lungs) equals approximately 1,000 ml. per square meter of body surface per day in the hospitalized patient. When respiration is hyperactive or fever is present, this loss may be greatly increased.

**Sweat losses (sensible perspiration) depend chiefly upon temperature, humidity, metabolic rate, and fever. In disease, they may equal 1,000 or more milliliters per square meter of body surface per day. Sweat losses can be roughly measured by weight changes if one considers other gains and losses of fluids.

It is, of course, important to recognize that several imbalances may be present in a given patient. With 12 specific fluid imbalances relating to the properties of extracellular fluid, two to its position and three to the nutritional status of the body, the number of possible combinations is great. Confusion can be minimized by careful use of the systematic approach.

Moyer points out that when two imbalances are present, identical signs produced by these imbalances are additive; opposing signs or findings will tend to neutralize each other. He gives as an example the tendency of the body temperature to fall with an extracellular fluid volume deficit not accompanied by change in total electrolyte concentration. This fall in temperature will be counterbalanced by the tendency of the temperature to rise should the electrolyte concentration be excessive. The result would be a normal temperature. He refers to such a neutralization as "an algebraic composite." In practice such composite pictures cause less difficulty in diagnosis than might be expected.

SUMMARY

1) Careful attention to clinical history, physical examination and laboratory findings is essential for the diagnosis of fluid imbalances.

2) In taking a clinical history of the patient with fluid imbalance, attention should be focused on events and diseases affecting the skin, central nervous system, cardiovascular system, lungs, gastrointestinal tract, kidneys and glands of internal secretion. Gains and losses of water and electrolytes revealed by the clinical history should receive special consideration.

3) Findings in the physical examination such as variations from the normal of sensorium, skin, mucous membranes, heart, blood vessels, lungs, stomach, intestines, kidneys, muscles, bones, nervous system, body temperature and body weight should be searched for methodically.

4) Abnormal laboratory findings are confirmatory rather than diagnostic.

15

Help From the Laboratory

The causes of all diseases are to be found in the blood.
HEBREW PROVERB

LIMITATIONS TO LABORATORY HELP

THERE are serious restrictions to the help which the laboratory can provide in the diagnosis and management of patients with fluid imbalances. Many practitioners have available only the simplest of laboratory procedures. Many others have the benefit of information given by more complex tests but must wait from two to eight hours for the laboratory report. When it arrives the time for decision has passed.

Even when the results of tests are immediately available, problems are encountered. Many test procedures have difficulties inherent in them which prevent consistently accurate results, even when the work is done by highly competent technicians. Obviously, the result of any test performed by an incompetent technician is worse than useless.

Still another problem looms large. We do not know the "normal" values for illness, since our tables of laboratory values are based on healthy individuals. It may sometimes be undesirable or even dangerous to attempt to restore the "abnormal" value of a sick patient to "normal."

If the laboratory is to provide maximal help, results of tests must be viewed as *confirmatory* rather than as *diagnostic*. The necessity for such a viewpoint is clearly seen when one recalls that the *plasma* potassium level may be elevated when there is actually a *cellular* potassium deficit. Values for the formed elements of the blood as the hemoglobin, hematocrit and red blood cell count are meaningless unless the patient's values prior to the onset of the imbalance are known.

Similarly, although the urine is ordinarily acidic in acidosis it may be alkaline even though acidosis is present in the following clinical conditions:

- Chronic renal infection in which the infecting organism converts urea in newly formed urine to ammonia,
- Milkman's syndrome, a severe chronic acidosis apparently secondary to renal tubular dysfunction,
- Fanconi's syndrome, a form of chronic acidosis secondary to renal tubular dysfunction,
- Sulfanilamide intoxication secondary to renal tubular dysfunction,
- Pharmacologic alkalinuria produced by a carbonic anhydrase inhibitor,
- Persistent acidosis secondary to renal tubular dysfunction in infants.

In all of these syndromes there is elevation of the plasma chloride and depression of the plasma bicarbonate.

Although the urine is usually alkaline in alkalosis, an acid urine may occur when alkalosis is accompanied by a severe potassium deficit. The mechanism of production of acid urine in this situation is not completely understood; it is believed to be related to retention of base bicarbonate induced by the potassium deficit. Acid urine in alkalosis is also favored by the administration of large amounts of sodium chloride solution in the management of this condition.

AVAILABLE TESTS

Most hospital laboratories are able to determine the plasma level of calcium, chloride, phosphorus, protein, albumin and bicarbonate. The flame photometer has made possible the accurate determination of the level of plasma sodium and plasma potassium. If a flame photometer is not available, one can, with certain limitations described previously, use the sum of the plasma chloride and the plasma bicarbonate plus 12 as a rough criterion of the plasma sodium level, provided all values are given in milliequivalents per liter. The hemoglobin is a fairly accurate index of the state of protein nutrition when iron deficiency anemia is not present.

Electrocardiographic tracings have been used by many in place of plasma potassium determinations. Which of the two criteria is more reliable as an index of the cellular stores of potassium is open to question. The present consensus is that both reflect the plasma potassium level, hence, indicate the level of potassium in the extracellular fluid.

The electrocardiograph also helps in the diagnosis of calcium deficit. Nor should the X-ray be forgotten as a diagnostic aid in fluid imbalances. It is of help in diagnosing errors in calcium metabolism, pituitary disease, renal concretions and certain vitamin deficits.

Several reliable tests, simple to perform, have been introduced by Scribner and Caillouette. The Rochester Bedside Lab® includes equipment, reagents and instructions needed for determination of total base (referred to in this text as total cations), chloride and bicarbonate of the plasma, by the Scribner Method. It is available from the Rochester Products Company, Rochester, Minnesota.

Microchemical determinations which can be performed on blood obtained by needle puncture represent a signal contribution. Such determinations are described by Graham and others in their article "Development of Neonatal Electrolyte Homeostasis" and by Natelson and others in their comprehensive manual *Correlation of Clinical and Chemical Observations in the Immature Infant.*

Even the physician with practically no formal laboratory facilities available can obtain some analytic help. The pH of the urine, so helpful in the differentiation of acidosis and alkalosis, can be simply performed at the bedside with the use of Nitrazine® paper. Urinary specific gravity, helpful in diagnosis of changes in volume and electrolyte concentration of extracellular fluid, is simply determined. Many physicians can have hemoglobins, hematocrits and red blood cell counts performed in their offices or clinics.

Variations in laboratory values for individuals of different ages must always be considered. The red blood cell count, hemoglobin, hematocrit and the plasma bicarbonate vary greatly between children and adults. In evaluating results of laboratory tests, both the average values and the normal range should be considered. These are presented in tables for convenient reference (see Tables 3 and 4).

Most plasma levels are best reported as milliequivalents per liter for reasons discussed in previous chapters. When they are reported in other terms, they can be quickly converted by reference to the conversion table which is presented inside the handbook covers.

MINIMAL DAILY PROGRAM

A minimal daily program for laboratory control of the patient with imbalance of the body fluids should include determination of the *plasma bicarbonate* and *plasma chloride* if facilities are available. Results of these tests reveal the effect of therapy on the electrolytes of the extracellular fluid. Initial determinations of the

TABLE 3

Laboratory Values

I. Normal Ranges

*A. Blood Formed Elements**

	Birth	*3 mo.*	*1 yr.*	*5 yr.*	*12 yr.*	*Women*	*Men*
RBC—million/cu. mm.	4.1-5.7	3.1-4.7	3.9-4.7	4.0-4.8	4.3-5.1	4.2-5.0	4.8-6.0
Hemoglobin—Gm./100 ml.	14-20	9-13	11-12.5	12-14.7	13.4-15.8	13-16	15-18
Hematocrit—% Vol. of packed RBC/100 ml.	43-63	28-40	32-40	36-44	39-47	39-47	44-52

B. Plasma Chemical Constituents

Plasma Na^+	137 – 147 mEq./L.
Plasma K^+	4.0 – 5.6 mEq./L.
Plasma Ca^{++}	4.5 – 5.8 mEq./L.
Plasma Cl^-	98 – 106 mEq./L.
Plasma Protein	6 – 8 Gm./100 ml.
Plasma HCO_3^-	Adults: 25–29 mEq./L. Children: 20–25 mEq./L.
Plasma Cl^- plus Plasma HCO_3^-	123 – 135 mEq./L.
Plasma pH	7.35–7.45
Plasma $HPO_4^=$	1.7 – 2.6 mEq./L.

C. Urine Values

Urine pH	4.5 – 8.2
Urine specific gravity	1.010 – 1.030

*Covers 94% of normal population.

TABLE 4

LABORATORY VALUES

II. AVERAGE VALUES IN HEALTH

A. Blood Formed Elements

	Birth	*3 mo.*	*1 yr.*	*5 yr.*	*12 yr.*	*Women*	*Men*
RBC—million/cu. mm.	4.9	3.9	4.3	4.4	4.7	4.6	5.4
Hemoglobin—Gm./100 ml.	17.1	11.1	11.7	13.3	14.5	14.5	16.5
Hematocrit—% Vol. of packed RBC/100 ml.	53	43	36	40	43	43	48

B. Plasma Chemical Constituents

Plasma Na^+	142 mEq./L.
Plasma K^+	5 mEq./L.
Plasma Ca^{++}	5 mEq./L.
Plasma Cl^-	103 mEq./L.
Plasma Protein	7.0 Gm./100 ml. (16 mEq./L.)
Plasma HCO_3^-	Adults: 27 mEq./L. Children: 23 mEq./L.
Plasma pH	7.4
Plasma $HPO_4^=$	2 mEq./L.

C. Urine Values

Urine pH	6.0
Urine specific gravity	1.015

hemoglobin, urinary pH, and *urinary specific gravity* are also recommended. If abnormal, these tests should be repeated daily until they return to normal. While *body weight* and *fluid intake-output measurements* are not laboratory procedures, they should be mentioned here since they provide important objective information which helps guard against gross overprovision or underprovision of fluids. When feasible they should be performed daily until fluid balance is achieved.

SUMMARY

1) Laboratory usefulness is restricted by:
   - unavailability of certain tests
   - time required to perform tests and send reports
   - technical problems inherent in tests
   - lack of knowledge concerning true normal values
   - misleading information sometimes provided by tests

2) Simple tests such as determination of the pH of the urine, specific gravity of the urine, volume of the urine, and blood hemoglobin should not be disregarded.

3) Electrocardiograph and x-ray may play important roles in diagnosis.

4) Simplified bedside tests and microchemical determinations offer great promise.

5) Laboratory values reported in *milligrams per 100 ml.* should be converted to *milliequivalents per liter,* using the table on the inside front and inside back cover.

Part III Therapy: Changes In Properties of Extracellular Fluid

16

Things to Come

For I dipt into the future, far as human eye could see, . . .

ALFRED, LORD TENNYSON

THE RECENT discovery of a great Mayan City, long lost in the trackless immensity of the jungle, was made possible by exploratory flights over thousands of square miles of primitive country. Armed with rough maps drawn from the vantage point of the sky, the indomitable Dana and Ginger Lamb made their way on foot to the fabulous lost metropolis of the Mayan Empire.

In the chapters to come, we shall present a simple, yet comprehensive, system of therapy of body fluid imbalances. Before proceeding on foot to our goal of understanding and employing this system, let us take a brief "aerial" glimpse at the shape of things to come later in the book.

Our first chapter on therapy will describe a logical, simple "yardstick" for dosage based on body surface area. We shall show

how this yardstick, which can be determined almost instantly from the height and weight, simplifies dosage.

The next six chapters describe a simple plan for managing most of the changes in properties of extracellular fluid and for maintenance of patients not suffering from fluid imbalance but dependent upon parenteral or oral fluids for water and electrolytes.

This plan is built around the so-called *balanced solutions.* When such solutions are given in doses that meet the *fluid volume needs* of the patient, they supply electrolytes in such quantities that the body homeostatic mechanisms can retain or reject them in accordance with the needs. To determine the daily dose for a given patient, the physician needs to know only the patient's surface area in square meters and the dosage level. The surface area is obtained from the height and weight. There are three levels of dosage based on the degree of fluid volume need. The appropriate dosage level, multiplied by the patient's surface area, gives the 24-hour dose.

In some patients, one or two auxiliary solution types may be indicated. Thus, for patients with temporary renal depression due to severe fluid volume deficit, a *pump priming solution* is given according to a simple dosage scheme to restore renal function. For patients who are losing body fluids abnormally, special *replacement solutions* are added to the daily fluid ration in volumes equal to the loss.

The plan described covers the great majority of fluid imbalances. It does not cover all. When body homeostasis is impaired, special measures must be taken, which are alluded to in Chapter 25. Certain imbalances involving urgent needs are not covered by the simplified plan. These include *deficit in total electrolyte concentration of extracellular fluid, calcium deficit, severe base bicarbonate deficit, severe base bicarbonate excess,* and *whole blood deficit.* Their precise management is described in Chapter 26.

The weighty problem of the burned patient, adequate subject for a volume in itself, is covered in a simple step-by-step manner in Chapter 27. Management of certain excesses, including *extracellular fluid volume excess, potassium excess* and *calcium excess,* is described in Chapter 28.

The therapy of changes in position of extracellular fluid is covered in the chapter "When Fluid Shifts Occur." The attention that should be given to nutritional deficits is the subject of Chapter 30. "Which Road to Scotland" presents routes of administration.

Variations in names applied by different manufacturers to their parenteral and oral solutions have made it necessary for us to employ either generally used or coined generic names in our discussions of fluid therapy. Representative commercial solutions corresponding to these generic names are presented in Table 28.

The proof of the pudding is in the eating, and Chapters 32 and 33 will enable the reader to determine for himself whether our pudding is digestible. . . .

17

Yardstick for Dosage

> *And he who spoke with me had a measure, a golden reed, . . .*
>
> Apocalypse

Proper dosage is of cardinal importance in fluid therapy, which so frequently involves parenteral administration of water, electrolytes, carbohydrate and, on occasion, whole blood, plasma, dextran, amino acids and vitamins. For proper dosage the physician requires a simple, usable and universally applicable dosage yardstick, a sort of twentieth century "golden reed."

DOSAGE CRITERIA IN USE

A combination of body weight and age is commonly used for determining dosage. This criterion has the advantage that age and weight are easily determined or approximated. It has one serious disadvantage when it is employed for determining dosages of water and electrolytes: requirements for these substances when expressed in terms of body weight vary greatly in different age groups. Thus, the young infant has an average water intake of 70 ml. per pound, while that of the 10-year-old is 35 ml. per pound and of the adult 25 ml. per pound. Similarly, the infant has an average intake of 1.3 mEq. of sodium per pound compared to the average intake of 0.8 mEq. per pound for the 10-year-old and 0.5 mEq. per pound for the adult.

An extremely useful dosage criterion—body surface area—has recently come into use in the field of parenteral fluid therapy. *Water and electrolyte requirements when expressed in terms of body surface area are approximately the same for all age groups.* This is a distinct advantage because the physician can employ surface area as a *universal dosage criterion.* Given the height and weight, body surface area can be determined in a few seconds by the use of nomograms.

It should be emphasized that body surface area values based on height and weight measurements represent rough approximations. Their employment does not constitute a method of determining dosage that is fundamentally different from the use of weight per se. Rather, use of body surface area as a dosage criterion is an expedient, a convenience which makes it possible for the physician to calculate dosage for persons of all ages, using one basic dosage schedule.

DETERMINATION OF BODY SURFACE AREA

Tables 5 and 6 are *body surface area nomograms.* It can be seen that they consist of three vertical scales. The left-hand scale indicates height in inches and centimeters. The right-hand scale indicates weight in pounds and kilograms. The middle scale indicates body surface area in square meters. To determine body surface area, draw a straight line between the points on the height and weight scales representing the patient's height and weight. The point at which this line crosses the surface area scale represents the patient's surface area in square meters. Thus, in a matter of seconds, the physician can determine the approximate surface area of any patient. *When determining the individual patient's body surface area, his normal or pre-illness weight should be used.*

REQUIREMENTS AND TOLERANCES IN TERMS OF BODY SURFACE AREA

A number of authors including Talbot and associates, Lowe, and Raile have published articles indicating the average maintenance requirements for water, sodium and potassium per unit of body surface area. Talbot and associates have also indicated the minimal needs and maximal tolerances for water, sodium and potassium in terms of body surface area. The minimal number of calories as carbohydrate necessary to minimize, although not completely eliminate, the ketosis and tissue breakdown of fasting has also been determined.

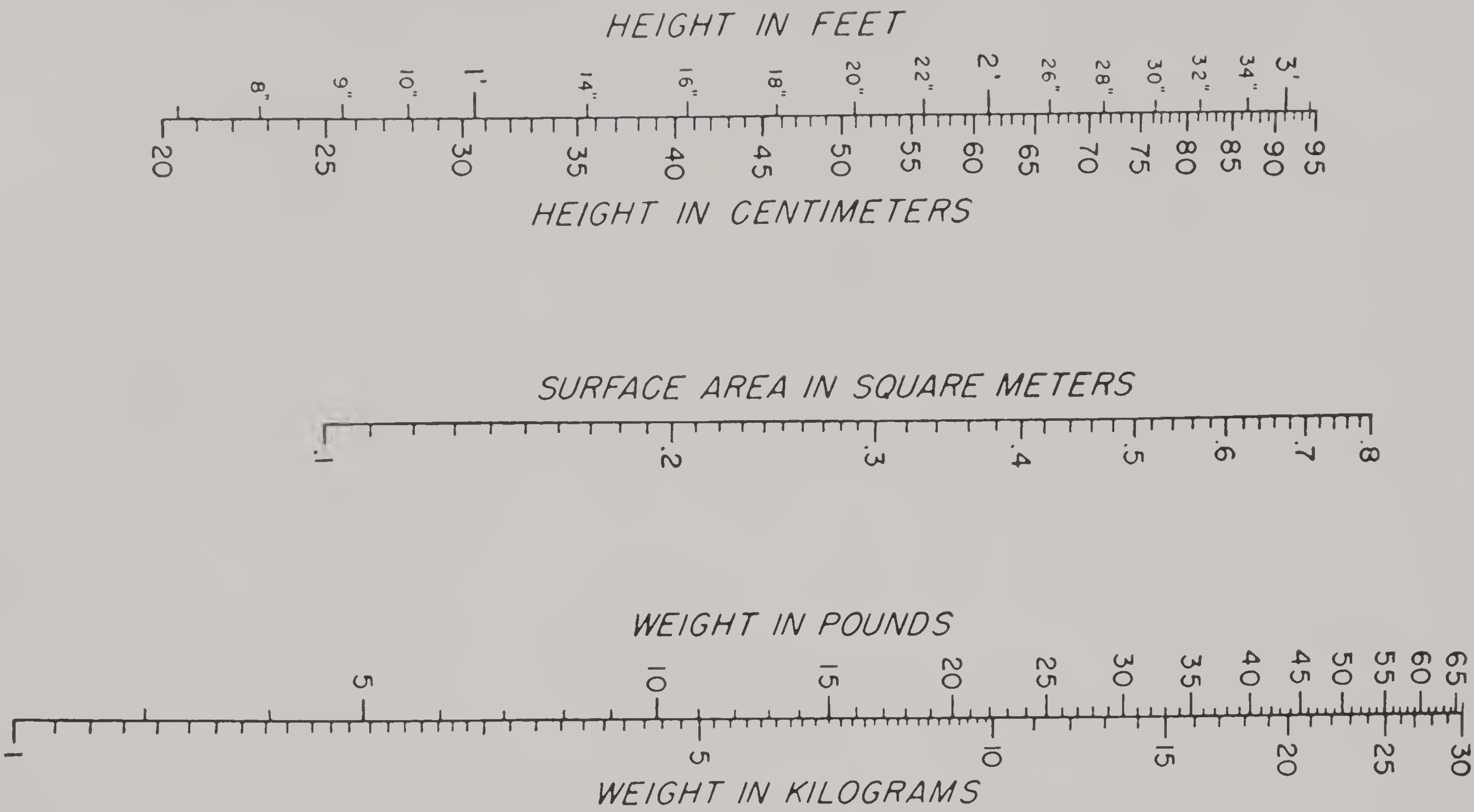

TABLE 5

Body Surface Area Nomogram for Infants and Young Children

To determine the surface area of the patient draw a straight line between the point representing his height on the left vertical scale to the point representing his weight on the right vertical scale. The point at which this line intersects the middle vertical scale represents the patient's surface area in square meters.

Used by permission of Talbot, N. B., Sobel, E. H., McArthur, J. W., and Crawford, J. D.: *Functional Endocrinology From Birth Through Adolescence*, Cambridge, Mass., The Commonwealth Fund, Harvard University Press, 1952.

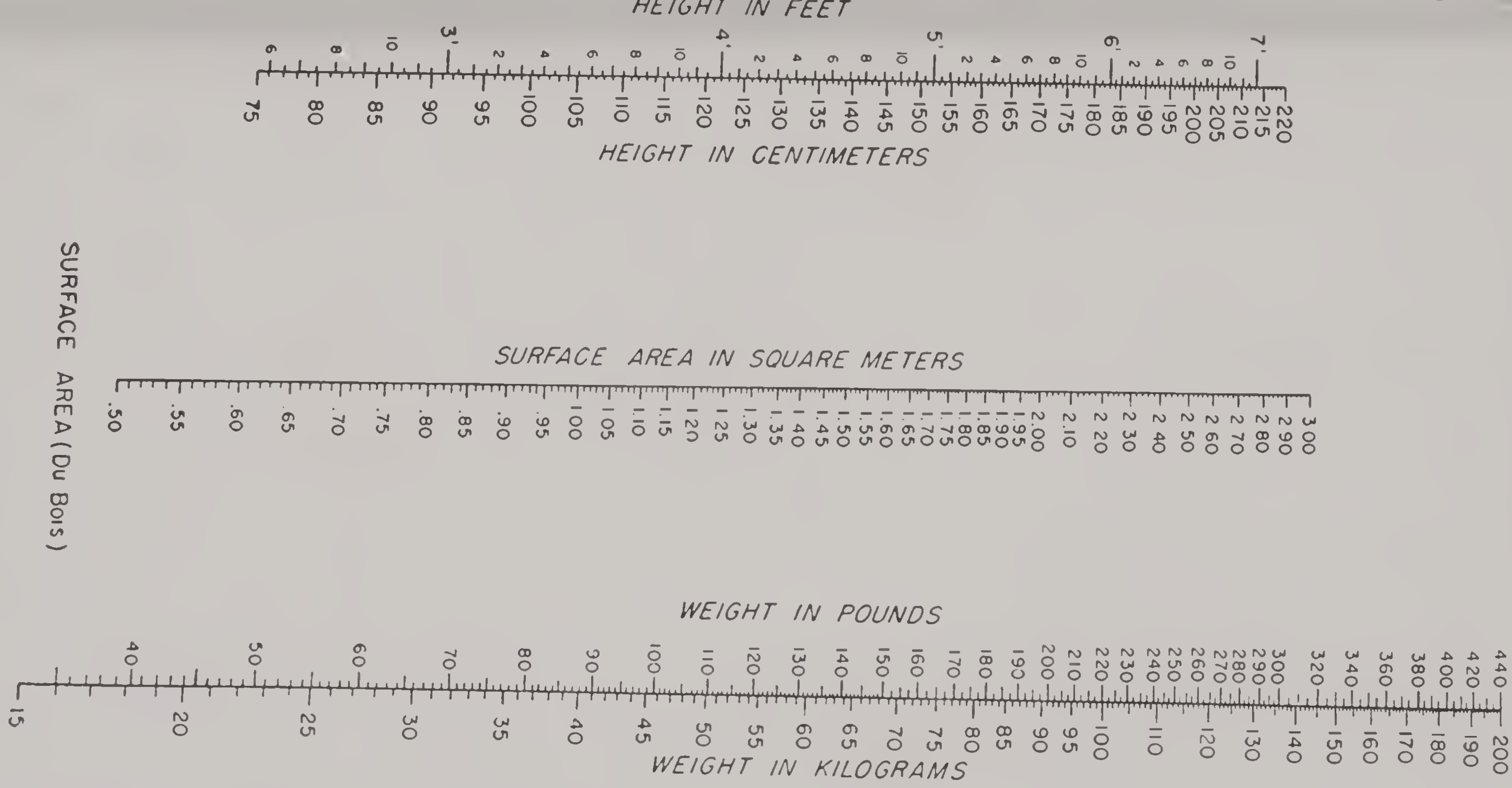

TABLE 6

Body Surface Area Nomogram for Older Children and Adults

To determine the surface area of the patient draw a straight line between the point representing his height on the left vertical scale to the point representing his weight on the right vertical scale. The point at which this line intersects the middle vertical scale represents the patient's surface area in square meters.

Used by permission of Talbot, N. B., Sobel, E. H., McArthur, J. W., and Crawford, J. D.: *Functional Endocrinology From Birth Through Adolescence*, Cambridge, Mass., The Commonwealth Fund, Harvard University Press, 1952.

The *average daily maintenance requirements* for water, sodium and potassium per square meter of body surface, as presented by these authors, are as follows:

water	1500 ml.
sodium	50 to 70 mEq.
potassium	50 to 70 mEq.

They have calculated, on the basis of work done by Gamble and Butler, that a daily carbohydrate intake of 60 to 75 Gm. per square meter of body surface will minimize the ketosis and tissue breakdown that occurs during fasting.

The 24-hour *minimal needs* and *maximal tolerances* of water, sodium and potassium per square meter of body surface are as follows:

	Minimal Needs	*Maximal Tolerances*
water	700 ml.	2700 ml.
sodium	10 mEq.	250 mEq.
potassium	10 mEq.	250 mEq.

The estimation of the minimal need for water is that for a normal, healthy individual. It takes into consideration water lost as insensible perspiration and as urine, as well as water gained as water of oxidation and from the breakdown of body tissue.

The estimation of the maximal tolerance for water is also that for a normal, healthy individual and indicates the highest level of water intake which is commensurate with normal electrolyte concentration of the extracellular fluid. Should the water intake exceed this level, an electrolyte concentration deficit will occur, because water is being taken into the body more rapidly than it can be excreted.

The major limiting factor for water tolerance is the ability of the kidney to form a dilute urine. The greatest amount of water that the kidney can excrete per unit (milliosmol) of dissolved substances is between 10 and 15 ml. When the amount of water to be excreted exceeds too greatly the dissolved substances to be excreted, water accumulates in the body.

It can be seen that the ranges between the minimal needs and maximal tolerances for sodium and potassium are also wide. However, recall that 2 liters of isotonic solution of sodium chloride contain a total of 308 mEq. of sodium, well in excess of the maximal tolerance per square meter of body surface per day for this cation.

SUMMARY

1) The most useful dosage criterion for parenteral fluid therapy is surface area, since water and electrolyte requirements per square meter of body surface are approximately the same for all age groups.

2) Given the patient's height and weight, the surface area can immediately be read from a nomogram.

3) Average daily maintenance requirements per square meter of body surface are:

water	1500 ml.
sodium	50 to 70 mEq.
potassium	50 to 70 mEq.
carbohydrate	60 to 75 Gm.

4) *Maintenance requirements* lie between *minimal needs* and *maximal tolerances.*

18

Balanced Solutions: Their Philosophy

> *In philosophy, it is not the attainment of the goal that matters, it is the things that are met with by the way.*
>
> HAVELOCK ELLIS

GREAT advances have been made in the understanding of body fluids both in health and disease during the past few years. The practicing physician shares the responsibility of transferring this new knowledge from the realm of the research laboratory to the treatment of patients with fluid imbalances. At times this transfer has been far from easy. Because of many factors, confusion has been widespread. Certainly, the great variety of complex regimes for the application of fluid therapy has contributed to the chaos and to a feeling of futility in the minds of many good physicians concerning the whole important subject of fluid balance.

The development of a single all-purpose solution useful for the treatment of all fluid imbalance states would be ideal. In the light of current knowledge, this ideal is unrealistic and may perhaps never be attained. Nevertheless, in recent years solutions have been developed which represent an immensely important step toward a single "all-purpose" solution. They are known variously as "multiple electrolyte solutions," "polyionic solutions" and "polyvalent solutions." Technically speaking, any electrolyte-containing solution could be designated by one of these names. For the purposes of this handbook, however, we have chosen to designate these solutions "balanced solutions," since they are so designed that *when used to meet the patient's water needs they supply electrolytes in amounts balanced between the minimal needs and the maximal tolerances.*

HISTORICAL DEVELOPMENT OF THE BALANCED SOLUTIONS

The *balanced solutions* stem largely from the thinking, research and clinical experiences of the pediatric group at the Massachusetts General Hospital led by Allan Butler and Nathan Talbot. Their students, both at the Massachusetts General Hospital and other institutions, have contributed further to the formulation and clinical use of these solutions.

At least two sets of observations apparently stimulated these investigators initially. First, they noted in the course of their studies that the composition of the fluids *retained* by patients recovering from diverse types of fluid imbalances often bore little resemblance to the composition of the fluids *administered*. For example, it was quite unusual for a patient to retain water, sodium and chloride in the proportions in which these substances are available in isotonic solution of sodium chloride.

Their second observation was that the body homeostatic mechanisms were ultimately responsible for the correction of fluid imbalances. Thus, it became apparent that every successful system of fluid therapy provides water and electrolytes in amounts which permit the body regulatory mechanisms to selectively utilize or discard them according to the body's requirements.

These investigators then surmised that solutions might be formulated which would be useful in a multitude of fluid imbalance states. The basic philosophy of such solutions is that, when given in quantities appropriate to meet the body's water needs, they will provide electrolytes in amounts that permit the body homeostatic mechanisms to selectively retain or reject these substances in accordance with body needs.

The functional integrity of the body homeostatic mechanisms is essential for the successful use of the *balanced solutions*. These homeostatic mechanisms were discussed in detail in Chapter 7.

SUMMARY

1) *Balanced solutions* are so designed that when used to meet the patient's water needs, they supply electrolytes in amounts *bal-*

anced between the minimal needs and the maximal tolerances for these substances.

2) In order for *balanced solutions* to be used successfully, the body homeostatic mechanisms must be functioning.

19

Balanced Solutions: Their Description

> *The young physician starts life with twenty drugs for each disease, and the old physician ends life with one drug for twenty diseases.*
>
> SIR WILLIAM OSLER

IT IS POSSIBLE to formulate a host of *balanced solutions* by slightly altering the amounts of incorporated electrolytes. In this handbook *four balanced solutions* will be described. These will include a general purpose *balanced solution* formulated by Butler and Talbot, a general purpose *balanced solution* formulated by Lowe, a special *balanced solution* formulated specifically for use in the newborn period, and an oral *balanced solution.*

NOMENCLATURE

Nomenclature is a problem with therapeutic fluids as it is with other agents. To refer to a specific *balanced solution* as "Butler's solution" or "Lowe's solution" is confusing, since there are several "Butler's solutions" and several "Lowe's solutions." Therefore, it is wise to employ another system for naming these solutions: we shall give each *balanced solution* a number which represents the sum in milliequivalents of the cations (or anions) present in a liter of that solution.

For example, the *balanced solution* devised by Butler and Talbot which we have employed, contains a total of 75 mEq. of cations (or anions) per liter of solution. It will be designated No. 75 *balanced solution.*

SOLUTION FORMULATED BY BUTLER AND TALBOT

No. 75 *balanced solution* has the following electrolyte composition for each liter of solution:

cations	sodium	40 mEq.
	potassium	35 mEq.
anions	lactate	20 mEq.
	chloride	40 mEq.
	phosphate	15 mEq.

Carbohydrate can be omitted or incorporated in amounts of 50 to 100 Gm. per liter of solution, that is, in 5 or 10% concentration.

Inspection of the solution reveals that it contains a total of 75 mEq. of cations and 75 mEq. of anions per liter.

SOLUTION FORMULATED BY LOWE

No. 90 *balanced solution* formulated by Lowe has the following electrolyte composition for each liter of solution:

cations	sodium	60 mEq.
	potassium	25 mEq.
	magnesium	5 mEq.
anions	chloride	53 mEq.
	lactate	25 mEq.
	phosphate	12 mEq.

Carbohydrate can be omitted or incorporated in amounts of 50 to 100 Gm. per liter of solution, that is, in 5 or 10% concentration.

Inspection of this solution reveals that it contains a total of 90 mEq. of cations and 90 mEq. of anions per liter.

SOLUTION FORMULATED FOR THE NEWBORN PERIOD

One *balanced solution* has been designed primarily for use in full term and large premature infants during the newborn period, that is, up to the end of the first month of life. It contains a smaller amount of electrolytes than either No. 75 *balanced solution* or No. 90 *balanced solution*. This lower electrolyte concentration takes into consideration the immaturity of the infant kidney.

This *balanced solution* has the following electrolyte composition for each liter of solution:

cations	sodium	25 mEq.
	potassium	20 mEq.
	magnesium	3 mEq.
anions	chloride	22 mEq.
	lactate	23 mEq.
	phosphate	3 mEq.

As with the other *balanced solutions,* carbohydrate can be omitted or added in amounts of 50 to 100 Gm. per liter of solution, that is, in 5 or 10% concentration.

Inspection of this solution reveals that it contains a total of 48 mEq. of cations (or anions) per liter. It will be referred to as No. 48 *balanced solution.*

BALANCED SOLUTION FOR ORAL ADMINISTRATION

An oral *balanced solution* formulated by Darrow and Cooke is useful in mild and moderate fluid imbalance states and during the recovery phase of severe fluid imbalances, provided oral fluids are tolerated and not contraindicated.

To prepare the oral *balanced solution,* 80 Gm. (eight fillings of the measure that is provided with the commercial preparation) of the electrolyte mixture, referred to in Figure 28, is dissolved in one quart of water. The resultant solution has the following electrolyte composition per liter:

cations	sodium	50 mEq.
	potassium	20 mEq.
	calcium	4 mEq.
	magnesium	4 mEq.
anions	citrate	30 mEq.
	lactate	4 mEq.
	chloride	30 mEq.
	sulfate	4 mEq.
	phosphate	10 mEq.

When used to meet the water needs of the patient, it provides electrolytes in amounts lying between the minimal needs and the maximal tolerances.

Each liter of oral *balanced solution* also provides 70 Gm. of carbohydrate. The solution provides a total of 78 mEq. of cations (or anions) per liter when the proper dilution is employed.

RATIONALE FOR CHLORIDE, LACTATE, PHOSPHATE AND MAGNESIUM

Chloride, lactate, phosphate and magnesium are incorporated in *balanced solutions* in physiologic quantities, to help meet needs without exceeding tolerances for these ions.

FREE WATER

The total electrolyte concentration of *balanced solutions* is about one-half of the electrolyte concentration of extracellular fluid or of solutions such as isotonic solution of sodium chloride, lactated Ringer's solution or Ringer's solution. Thus, the extracellular fluid has 150 mEq. of cations (or anions) per liter as contrasted with 75 mEq. per liter in No. 75 *balanced solution*, 90 mEq. per liter in No. 90 *balanced solution*, 48 mEq. per liter in No. 48 *balanced solution* and 78 mEq. per liter in the oral *balanced solution*.

Thus, these solutions contain about twice as much water per unit of electrolyte as does extracellular fluid. Since any carbohydrate present in the solution is quite rapidly metabolized, these solutions will provide extra or free water to the body. We use the term "free" in the physiologic rather than the chemical sense. Clearly, water is not free, that is, available to meet other needs of the body, when it must be used to maintain the normal proportion of water and electrolytes in the extracellular fluid. Thus, isotonic solution of sodium chloride provides *no* free (or available) water. A solution of *half* isotonic strength provides free water in an amount equal to *half* its volume.

Free water is useful to the body for two reasons: First, it replaces the essentially electrolyte-free water lost as insensible perspiration from the lungs and skin. Recall that normally about one-half of the water lost from the body each day is lost via this route.

Second, there is evidence that the normal kidney under stress may not be able to concentrate urine as well as it does during health. The free water which *balanced solutions* provide is available to aid the kidney under stress to perform adequately its excretory and regulatory functions.

RATIONALE FULFILLED

Inspection of all of these solutions reveals that when given at a dosage level of 1500 ml. per square meter of body surface, maintenance amounts of water and electrolytes will be provided. Either 50, 70 or 100 Gm. of carbohydrate incorporated in each liter of solution will provide the minimal amount of carbohydrate necessary to counteract the ketosis and tissue breakdown of fasting.

Fortunately, the usefulness of the *balanced solutions* does not stop with provision of water and electrolytes for maintenance. The great majority of body fluid imbalances involve an extracellular fluid volume deficit plus one or more deficits in electrolyte composition. Their treatment requires administration of greater-than-maintenance amounts of water and electrolytes. The *balanced solutions* are so formulated that when used to correct a fluid volume deficit they provide electrolytes in such quantities that the homeostatic mechanisms can:

1) Retain those required for normalization of body fluid electrolyte composition,
2) Excrete electrolytes which are not needed.

At the same time they provide carbohydrate to combat ketosis and tissue breakdown.

Thus, the *balanced solutions* are suitable for therapy of most body fluid imbalances, within limitations which will be described fully in subsequent chapters.

SUMMARY

1) There are four *balanced solutions:*

a) No. 75 *balanced solution*, containing 75 cations (or anions) per liter, is designed for parenteral administration to older infants, children and adults.

b) No. 90 *balanced solution*, containing 90 cations (or anions) per liter, is designed for parenteral administration to older infants, children and adults.

c) No. 48 *balanced solution*, containing 48 cations (or anions) per liter, is designed for parenteral administration to full term and large premature infants under 1 month of age.

d) Oral *balanced solution,* containing 78 cations (or anions) per liter, is designed for oral administration to all age groups. It is the *balanced solution of choice when oral administration is feasible.*

2) *Balanced solutions* have a far lower electrolyte concentration than extracellular fluid. Thus, they provide free or available water, which can replace water lost as insensible perspiration and which can aid the kidney under stress in performing its regulatory and excretory functions.

3) When a balanced solution containing carbohydrate in 5 or 10% amounts is given at a dosage level of 1500 ml. per square meter of body surface per day, it provides maintenance amounts of water, electrolytes and carbohydrate.

4) *Balanced solutions* can also be employed for the treatment of many body fluid imbalances which require greater-than-maintenance quantities of water, electrolytes and carbohydrate.

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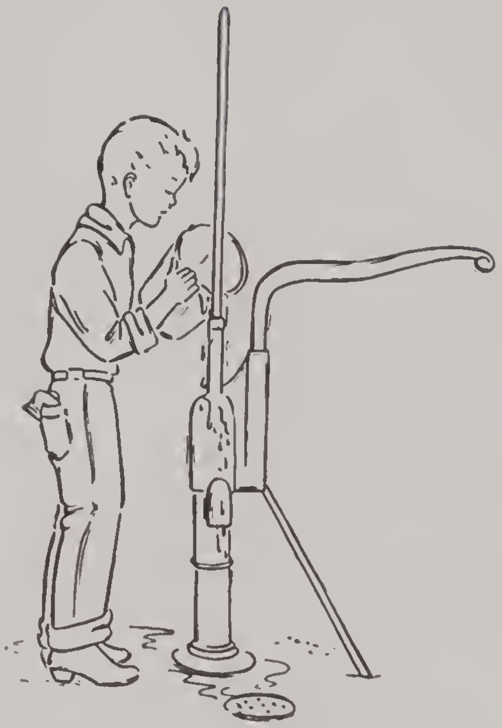
Pump Priming Solutions: Their Description



Before . . . the golden fillet shrink back, and the pitcher be crushed at the fountain, . . .

ECCLESIASTES

SINCE the four *balanced solutions* contain important amounts of potassium, a potentially toxic ion, they should not be administered until the homeostatic mechanisms of the body, particularly those controlled by the kidney, are known to be functioning at a level at which the electrolyte content of the extracellular fluid can be maintained within safe limits. It is recognized that the solutions will be used in many patients whose potentially normal kidney function is depressed because of body fluid volume deficits. Such reduction of kidney function is a protective mechanism designed to conserve as much body fluid as possible. It is unaccompanied by anatomic alterations in the kidneys.

As this type of renal depression develops, there first occurs an increase in urinary specific gravity. This is followed by a decrease in the volume of urine excreted. The following findings are evidence that frank renal depression is present:

1) Urinary specific gravity above 1.030.
2) Oliguria, evidenced by a history of voiding less than three times during the previous 24 hours.
3) Anuria, shown by absence of urine in the bladder.

Frank renal depression may be assumed to be present when there has been a recent fluid loss of great magnitude, such as occurs with severe infectious diarrhea of explosive onset.

Renal depression caused by a fluid volume deficit should be differentiated from acute renal insufficiency caused by shock, intravascular hemolysis, severe trauma, or the absorption of renal toxins such as the heavy metals. The clinical manifestations of the two types of depression of renal function are similar; history and management differ greatly.

INITIAL HYDRATING SOLUTION A PUMP PRIMER

In order to increase kidney function to the point where it can regulate the electrolyte composition of extracellular fluid, it is necessary to administer an initial hydrating solution which is free of potassium. The use of such a solution may be loosely compared to priming a well pump. In order to get the pump to work, it is first necessary to prime it by adding some water to the system. Likewise, in fluid volume deficits, it may be necessary to "prime" the kidney with a potassium-free solution to re-establish urinary flow. We call the solutions used for this purpose *pump priming solutions.*

TYPICAL PUMP PRIMING SOLUTIONS

Such solutions have about one-third to one-half the electrolyte concentration of extracellular fluid. They usually have 5 or 10% carbohydrate incorporated. Two solutions will be described.

The first *pump priming solution* has the following electrolyte composition per liter of solution:

sodium	51 mEq.
chloride	51 mEq.

It can be seen that this solution is simply a one-third isotonic solution of sodium chloride. We designate it No. 51 *pump priming solution* since it has 51 mEq. of cations (and of anions) per liter.

The second *pump priming solution* has the following electrolyte composition per liter of solution:

sodium	70 mEq.
chloride	55 mEq.
lactate	15 mEq.

We designate it No. 70 *pump priming solution* because of its 70 mEq. of cations (and of anions) per liter.

SUMMARY

1) The *balanced solutions* contain potassium, an essential though potentially toxic ion. These solutions should not be administered unless the homeostatic mechanisms of the body are functioning.

2) In many patients, kidney function is depressed solely because of an extracellular fluid volume deficit.

3) Renal depression is indicated by urinary specific gravity above 1.030, oliguria or anuria.

4) Administration of a *pump priming solution* to patients with depressed renal function enables the physician to:

   a) Restore the kidneys to normal function, if the cause of the depression is extracellular fluid volume deficit, *or*

   b) Discover that the renal depression is not the result of a fluid volume deficit but rather of serious renal impairment.

5) *Pump priming solutions* have about one-half the electrolyte concentration of extracellular fluid. There are two such solutions:

   a) No. 51 *pump priming solution*, potassium-free and containing 51 mEq. of cations (or anions) per liter, and

   b) No. 70 *pump priming solution*, potassium-free and containing 70 mEq. of cations (or anions) per liter.

21

Replacement Solutions: Their Description

> *One can weigh the secretions in the balance and measure the work of the heart in footpounds.*
>
> SIR WILLIAM OSLER

GASTROINTESTINAL REPLACEMENT SOLUTIONS

GASTROINTESTINAL REPLACEMENT SOLUTIONS are solutions used to replace abnormal losses of water and electrolytes through such routes as vomiting, gastric or intestinal suction, intestinal or colonic fistulas, enterostomy, cecostomy and colostomy. Replacement of such "concurrent losses" constitutes a facet of therapy which is distinct from providing maintenance amounts of water and electrolytes and from correcting deficits. Concurrent losses require replacement solutions whose concentration and composition approximate those of the fluid lost. Such are the *gastrointestinal replacement solutions.*

The *gastrointestinal replacement solutions* are roughly isotonic with extracellular fluid, that is, they contain approximately the same number of cations and anions per liter as does extracellular fluid. They do not supply the free water necessary for replacing insensible losses of water through the lungs and skin and for the urinary excretion of waste products.

The *gastrointestinal replacement solutions* which we shall discuss include one solution appropriate for replacement of gastric juice and three solutions suitable for replacement of intestinal secretions. In naming the *gastrointestinal replacement solutions,* we shall use the system employed for the *balanced solutions,* that is, we will give each solution a number equal to the total number of cations (or anions) in a liter of the solution.

Gastric Replacement Solution

This solution, No. 150 *gastrointestinal replacement solution,* was devised by Cooke and Crowley for use in the replacement of secretions lost through vomiting or gastric suction. Its formulation takes into account the relatively high chloride content of gastric juice. It is usually given in a form which provides either 5 or 10% carbohydrate (see Table 7). In order for this solution to supply an excess of chloride over sodium, there are 70 mEq. of ammonium in each liter. Ammonium is metabolized to urea in the liver.

Intestinal Replacement Solutions

There are two excellent intestinal replacement solutions, one designed by Lowe and one by Fox. These are designated No. 130 *gastrointestinal replacement solution* and No. 158 *gastrointestinal replacement solution,* respectively. Either is suitable for replacement of fluid losses through duodenal or intestinal suction, intestinal or colonic fistulas, enterostomy, cecostomy or colostomy.

No. 130 *gastrointestinal replacement solution* is designed primarily for replacement of fluids rich in potassium, as from a recent ileostomy or cecostomy. No. 158 *gastrointestinal replacement solution* is particularly useful for replacing losses of duodenal secretions. Lactated Ringer's solution is a fairly good general purpose intestinal replacement solution. However, it has the disadvantage of being relatively low in potassium.

Any of the intestinal replacement solutions can have 5 or 10% carbohydrate incorporated in them to help provide the additional calories which patients on parenteral fluid regimes invariably require.

Table 7 shows the compositions of the gastrointestinal secretions and of the *gastrointestinal replacement solutions.* Of particular interest is the fact that only gastric juice has an important content of hydrogen. It is this high concentration of hydrogen that accounts for the extreme acidity of gastric juice.

REPLACEMENT OF STOOLS AND SWEAT

Concurrent losses of hypotonic fluids such as perspiration and watery stools of diarrhea are best replaced by approximately equal

TABLE 7

GASTROINTESTINAL SECRETIONS

Average Electrolyte Content in Milliequivalents per Liter

	Cations			*Anions*	
	Hydrogen	*Sodium*	*Potassium*	*Bicarbonate*	*Chloride*
Gastric juice	72	70	5	6	141
Pancreatic juice	—	144	5	116	33
Hepatic duct bile	—	144	5	40	109
Jejunal secretions	—	140	5	29	116

GASTROINTESTINAL REPLACEMENT SOLUTIONS

Electrolyte Content in Milliequivalents per Liter

	Cations					*Anions*			
	Sodium	*Potassium*	*Calcium*	*Magnesium*	*Ammonium*	*Lactate*	*Acetate*	*Citrate*	*Chloride*
No. 150 Gastrointestinal replacement solution (Cooke and Crowley)	63	17	—	—	70	—	—	—	150
No. 158 Gastrointestinal replacement solution (Fox)	140	10	5	3	—	—	47	8	103
No. 130 Gastrointestinal replacement solution (Lowe)	100	30	—	—	—	55	—	—	75
Lactated Ringer's solution (Hartmann)	130	4	3	—	—	28	—	—	109

volumes of a *balanced solution.* The quantity of *balanced solution* given should be in addition to that provided for meeting maintenance needs and correcting deficits.

SUMMARY

1) Losses of water and electrolytes through such routes as vomiting, gastric or intestinal suction, intestinal or colonic fistulas, enterostomy, cecostomy, colostomy, abnormal sweating or diarrhea are termed *concurrent losses.*

2) With the exception of losses from abnormal sweating and diarrhea, these losses are of approximately isotonic fluid, and should be replaced volume for volume with a *gastrointestinal replacement solution.*

3) No. 150 *gastrointestinal replacement solution* is intended for replacement of gastric secretions; No. 130 *gastrointestinal replacement solution* or No. 158 *gastrointestinal replacement solution* is useful for replacing losses of fluid from the small or large intestines; lactated Ringer's solution is useful for replacement of losses of intestinal secretions which are not especially high in potassium.

4) Losses from abnormal sweating or diarrhea are usually of a hypotonic fluid, and should be replaced volume for volume with a *balanced solution.*

22

Priming, Balanced and Replacement Solutions: Their Dosage

Antidotes are poison.
William Shakespeare

The physician should approach a fluid balance problem in the same fashion as he approaches any other clinical problem. He has three sources of information, namely, history, physical examination and laboratory determinations. From each of these he gathers valuable information to guide him in the formulation of therapy. He should remember in treating any patient with a fluid balance problem that the chief purpose of fluid therapy is the provision of water and electrolytes which:

1) Meet current maintenance needs.
2) Correct deficits.
3) Replace concurrent losses.

PUMP PRIMING SOLUTIONS

The potassium-free initial hydrating solution which we designate a *pump priming solution* is not a *balanced solution.* However, its use is indicated prior to the administration of *balanced solutions* in patients whose renal function is depressed because of fluid volume deficits.

The dose of the *pump priming solution* is 360 ml. per square meter of body surface in 45 minutes. Voiding indicates that kidney function is at an adequate level of efficiency. Most individuals with functional kidneys will void before the 45-minute period has passed.

If the patient has not voided by the end of 45 minutes, the dose of the *pump priming solution* is decreased to 120 ml. per square meter of body surface in 60 minutes. If the patient does not void within this 60-minute period, it is probable that his kidney function is severely impaired. Careful re-evaluation of the entire clinical situation is in order in such instances. Attention must be focused primarily on the renal dysfunction (see Chapter 25).

The amount of *pump priming solution* to be administered *when renal depression is present* is calculated as follows:

1) Determine patient's surface area in square meters from his height and weight by the use of surface area nomograms.
2) Multiply the dosage level of 360 ml. by patient's surface area in square meters. The result gives the amount of *pump priming solution* to be administered in the first 45 minutes.
3) If a *pump priming solution* is still necessary after 45 minutes, multiply 120 ml. by patient's surface area in square meters. This result gives the amount of *pump priming solution* to be administered during the next hour.

The rate of administration of *pump priming solution* can be expressed as milliliters per minute or drops per minute. *Milliliters per minute* will be used in this handbook because of variation in the number of drops contained in a milliliter when different types of dripmeters are used.

The recommended rate of administration of *pump priming solution* in milliliters per minute during the first 45 minutes is 8 ml. per square meter of body surface per minute (360 ml. divided by 45 minutes). To determine the rate for a given patient, multiply the patient's surface area in square meters by 8 ml.

The recommended rate of administration of *pump priming solution*, should it be necessary during the next hour, is 2 ml. per square meter of body surface per minute (120 ml. divided by 60 minutes). To determine the rate, multiply the patient's surface area in square meters by 2 ml.

BALANCED SOLUTIONS

A *balanced solution* is employed to provide water and electrolytes to meet current maintenance needs and to correct deficits. In addition, the *balanced solution* is ideal for replacing concurrent losses such as occur with excessive perspiration and watery diarrhea, as described in the chapter on *replacement solutions.* Let us here consider the dose of *balanced solution* for *meeting maintenance needs* and for *meeting maintenance needs plus correcting deficits.*

Dosage

Maintenance needs will be met by an intake of 1500 ml. of *balanced solution* per square meter of body surface per day. This amount would be used in patients who have no fluid volume deficit at the time parenteral fluid therapy is started, or in patients whose fluid volume deficit has been corrected by past therapy. Typical examples include fasting, preoperative or postoperative patients and patients who have undergone several days of parenteral fluid therapy and who must still be maintained by the parenteral route.

There are two dosage levels to supply the water and electrolytes for meeting *maintenance needs* plus *correcting deficits.* They correspond to clinical estimations of deficits in *body fluid volume:*

1) 2400 ml. of a *balanced solution* per square meter of body surface per day is given to meet maintenance needs and correct a *moderate* fluid volume deficit, and
2) 3000 ml. of a *balanced solution* per square meter of body surface per day is given to meet maintenance needs and correct a *severe* fluid volume deficit.

Recall that a moderate fluid volume deficit in an infant or small child is indicated by a weight loss up to 10% and a severe fluid volume deficit by a weight loss of more than 10%. In the larger child and adult, a 5% loss in body weight represents a moderate fluid volume deficit and more than 5% a severe fluid volume deficit. In addition to weight changes, all other evidence of fluid volume deficit including pertinent data from the clinical history, physical examination and laboratory tests should be employed in determining the dosage level.

Calculation of Daily Dose

The amount of *balanced solution* needed by a given patient during a 24-hour period is calculated as follows:

1) Determine the patient's surface area in square meters from his height and weight by use of surface area nomograms.

2) Multiply daily dosage level selected (1500, 2400 or 3000 ml.) by patient's surface area in square meters.

3) If a *pump priming solution* has been used, subtract the volume administered from the calculated dose for *balanced solution.*

Rate of Administration

The recommended rates of administration when a *balanced solution* is used to meet maintenance needs are:

1) 2 ml. per square meter of body surface per minute when the daily dose is administered as a *single infusion,* or

2) 4 ml. per square meter of body surface per minute when the daily dose is administered in *two infusions.* If this rate is used, the 24-hour parenteral fluid ration is divided into two separate infusions which are started 12 hours apart.

The rate of administration in milliliters per minute for a given patient when a *balanced solution* is used to meet maintenance needs is calculated as follows:

1) Multiply the patient's surface area in square meters by 2 ml. if the solution is administered as a *single infusion.*

2) Multiply the patient's surface area in square meters by 4 ml. if the solution is administered in *two infusions.* For example, a patient with 2 square meters of body surface required 3000 ml. of *balanced solution* for maintenance (1500 ml. x 2 square meters). This would be divided into two infusions of 1500 ml. each. One infusion would be given at the rate of 8 ml. per minute (4 ml. x 2 square meters) in approximately three hours (1500 ml. divided by 8 ml. per minute = 187 minutes) beginning at 8 a.m. and the other at the same rate in three hours beginning at 8 p.m.

The recommended rate of administration when a *balanced solution* is used to meet maintenance needs and correct a fluid volume deficit, either moderate or severe, is 3 ml. per square meter of body surface per minute. This rate, in milliliters per minute for a given patient, is calculated as follows:

Multiply the patient's surface area in square meters by 3 ml.

Patients who tolerate oral fluids can be given oral *balanced solution* in place of an equal amount of a parenteral *balanced solution.* The daily dose for the oral *balanced solution* is the same as for parenteral *balanced solutions.* The oral *balanced solution* is usually given ad libitum, however.

REPLACEMENT SOLUTIONS

Abnormal losses occurring simultaneously with therapy should be measured as accurately as possible. They should be replaced volume for volume by a *gastrointestinal replacement solution* or by additional amounts of a *balanced solution.* The former is indicated for replacement of significant losses of approximately isotonic fluids such as gastric or intestinal secretions. The latter should be used for replacement of losses of hypotonic fluids such as excessive perspiration or watery diarrhea.

For example, if it is estimated that 1000 ml. of ileostomy fluid is being lost through the day then 1000 ml. of the appropriate *gastrointestinal replacement solution* is given. If there is a concurrent loss of 500 ml. of diarrheal fluid, then 500 ml. of a *balanced solution* should be added to the day's ration.

While it is ideal to assess concurrent losses every eight hours and replace them in the following eight hours, this interval is frequently impractical; hence, we use a 12 or even 24-hour interval for assessment of concurrent losses.

The recommended rate of administration when a *gastrointestinal replacement solution* or *balanced solution* is used to replace a concurrent loss is 3 ml. per square meter of body surface per minute. This rate, in milliliters per minute for a given patient, is calculated as follows:

Multiply the patient's surface area in square meters by 3 ml.

Slower rates of infusion may be used if desired, so long as the daily parenteral fluid ration is administered in 24 hours. For example, slower rates are often used in infants and small children when parenteral fluid therapy is necessary for more than 24 hours because of technical difficulties associated with repeated venipunctures.

SUMMARY

1) The following simple dosage rules (which should be memorized) suffice for managing the great majority of fluid imbalance states:

a) *Pump priming solutions:* give 360 ml. per square meter of body surface in 45 minutes if renal function is depressed. If voiding occurs, proceed with *balanced solution.* If voiding does not occur, give 120 ml. of *pump priming solution* per square meter of body surface in 60 minutes. If voiding does not then occur, serious renal impairment is probably present (see Chapter 25). If voiding occurs, proceed with *balanced solution.*

b) *Balanced solutions:* there are three levels of dosage

1) *1500 ml.* per square meter of body surface covers *maintenance needs* for water and electrolytes.

2) *2400 ml.* per square meter of body surface covers *maintenance needs plus correction of moderate fluid volume deficits.*

3) *3000 ml.* per square meter of body surface covers *maintenance needs plus correction of severe fluid volume deficits.*

c) *These doses are per 24-hour day.* The volume of *pump priming solution* administered should be subtracted from the calculated dose for *balanced solution.*

d) In addition to meeting maintenance needs and correcting deficits, if any, concurrent losses, if any, should be replaced. A *gastrointestinal replacement solution* should be added to the 24-hour parenteral fluid ration in a volume equal to losses of gastric or intestinal secretions. Additional *balanced solution* should be added to the 24-hour parenteral fluid ration in a volume equal to losses from abnormal perspiration or watery diarrheal stools.

2) The following are the recommended rates for intravenous administration of *pump priming solutions, balanced solutions* and *replacement solutions* provided body homeostasis is normal:

a) *Pump priming solutions:* 8 ml. per square meter of body surface per minute during the first 45 minutes. If voiding does not occur, 2 ml. per square meter of body surface per minute for the next 60 minutes.

b) *Balanced solutions:*

1) 2 ml. per square meter of body surface per minute when administered as a *single infusion* to meet *maintenance* needs, or

2) 4 ml. per square meter of body surface per minute when administered in *divided doses* to meet *maintenance* needs.

3) 3 ml. per square meter of body surface per minute when used:

a) to meet *maintenance needs* and *correct fluid volume deficits* (moderate or severe), or

b) to *replace concurrent losses.*

c) *Gastrointestinal replacement solutions:* 3 ml. per square meter of body surface per minute.

3) Slower rates may be employed as long as the daily parenteral fluid ration is given within 24 hours.

4) For patients able to ingest fluids, oral *balanced solution* can be given in place of a parenteral *balanced solution.*

23

Treatment of Most Changes in Properties of Extracellular Fluid

. . . I injected one hundred and twenty ounces,
when like the effects of magic,
instead of the pallid aspect of one
whom death had sealed as his own,
the vital tide was restored, and
life and vivacity returned!

THOMAS LATTA

FROM THE standpoint of incidence, the imbalances which involve changes in the properties of extracellular fluid are overwhelmingly the most important. In this chapter we shall outline a simple effective plan for the management of most changes in properties of extracellular fluid, based on principles developed by Butler, Talbot and associates and by Lowe and involving use of *priming, balanced* and *replacement solutions* as described in Chapter 22.

The changes in properties of extracellular fluid which are amenable to the simple plan of therapy here described include *fluid volume deficit, excess in total electrolyte concentration, potassium deficit, base bicarbonate deficit* and *base bicarbonate excess.* In addition, this plan of therapy can be used to support the compensatory efforts of the kidney in *carbonic acid deficit* and *carbonic acid excess.* It is apparent that this group of imbalances includes extracellular fluid volume deficit and several imbalances in electrolyte content. Any one imbalance or varying combinations may be present in a given patient. In either case, the *balanced solutions* can be employed for treatment.

In most patients a fluid volume deficit is combined with one or more of the electrolyte imbalances. In employing *balanced solutions* for management one uses the fluid volume need as a *gauge* for dosage. Let us suppose that a fluid volume deficit develops, either alone or accompanied by one or more electrolyte imbalances. The *balanced solution* is given at the dosage level appropriate to correct the fluid volume deficit, 2400 ml. per square meter of body surface per day for a moderate deficit or 3000 ml. for a severe deficit. Simultaneously, electrolytes are provided by the *balanced solution* within a range that permits the body to retain those needed and excrete those not required. Both the fluid volume deficit and any electrolyte imbalances present are corrected.

Should one or more electrolyte imbalances be present unaccompanied by a fluid volume deficit, the *balanced solution* is given at the level of 1500 ml. per square meter of body surface per day to satisfy the maintenance need for water. It will at the same time provide electrolytes within a range that permits the body to retain those needed and excrete those not required. The electrolyte imbalance is corrected, thanks to the selective action of the body homeostatic mechanisms.

Either the No. 75 *balanced solution* or the No. 90 *balanced solution* should be used for older infants, children or adults. In our clinical experience these two solutions are equally effective. They maye be regarded as *clinically interchangeable*. The No. 48 *balanced solution* is indicated for full term and large premature infants less than 1 month of age. Oral *balanced solution,* if tolerated, can be used in any age group. If adequate renal flow is not established, a *pump priming solution* should be administered, as previously described. The quantity of the *pump priming solution* given should be subtracted from the calculated dose for the *balanced solution.*

Concurrent losses of approximately isotonic fluids, such as gastric or intestinal secretions, should be replaced volume for volume by the appropriate *gastrointestinal replacement solution.* Concurrent losses of hypotonic fluids, such as perspiration or watery diarrheal stools, should be replaced volume for volume by the proper *balanced solution.* Replacement of concurrent losses, whether by *gastrointestinal replacement solution* or by *balanced solution*, is in addition to the amount of *pump priming solution* and *balanced solution* given to supply maintenance needs and correct deficits.

Before employing the simple plan just outlined, the physician should rule out fluid imbalances requiring *other* or *additional* therapy. These include conditions in which the body homeostatic mechanisms are not functioning, imbalances characterized by urgent needs, severe burns, certain excesses, changes in position of the extracellular fluid, and changes in the nutritional status of the body. These are fully discussed in chapters 24 through 30.

EXTRACELLULAR FLUID VOLUME CHANGES

Volume Deficit

This condition may consist of a decrease in the extracellular fluid volume without significant change in electrolyte concentration or composition. Frequently, however, a volume deficit is associated with a disturbance in extracellular electrolyte concentration or composition. Furthermore, deficits of extracellular fluid volume are often combined with deficits of cellular fluid volume, particularly when the former have existed for several days.

The correction of a fluid volume deficit consists of the provision of water and electrolytes in amounts that will restore the extracellular fluid to its normal volume. It is important to accomplish this without producing deficits or excesses in electrolyte concentration or composition. A solution containing water and electrolytes in the proportions present in extracellular fluid might appear to be the logical choice for treatment. However, such a solution would not provide the water necessary for the replacement of electrolyte-free water lost from the skin and lungs and for the excretion of metabolic waste products.

The *balanced solutions,* on the other hand, do provide the free water necessary for insensible losses and for the formation of urine. In addition, they provide the water and electrolytes necessary for the correction of the fluid volume deficit.

Volume Excess

The *balanced solutions* are not indicated for the treatment of volume excess. The treatment of this imbalance will be discussed in Chapter 27.

EXTRACELLULAR FLUID CONCENTRATION CHANGES

Deficit in Total Electrolyte Concentration

The *balanced solutions* are not indicated for the treatment of electrolyte concentration deficit. The treatment of this imbalance will be discussed in Chapter 26.

Excess in Total Electrolyte Concentration

Under normal conditions each 1000 ml. of extracellular fluid contains about 150 mEq. of cations and 150 mEq. of anions. There are about 3.3 ml. of water for each milliequivalent of electrolyte. In an extracellular fluid electrolyte concentration excess, this normal ratio of water to electrolyte is altered so that there are less than 3.3 ml. of water for each milliequivalent of electrolyte. This change can result from any clinical condition in which electrolytes accumulate in the extracellular fluid more rapidly than water, or in which water is lost from the extracellular fluid more rapidly than are electrolytes.

The correction of this imbalance consists of the provision of the water necessary to re-establish the normal water-to-electrolyte ratio in the extracellular fluid. A carbohydrate-in-water solution might seem to be the logical therapeutic choice. However, this imbalance is frequently associated with an extracellular fluid volume deficit which represents a loss of both water and electrolytes. Furthermore, electrolytes will be lost in the urine when its flow is established. Thus, maintenance amounts of electrolytes should be administered along with the water.

The *balanced solutions* provide the free water which will permit the re-establishment of the normal water-to-electrolyte ratio in the extracellular fluid. They also provide maintenance amounts of electrolytes.

EXTRACELLULAR FLUID COMPOSITION CHANGES

Potassium Deficit

Potassium is the major cellular cation and is present in the cell in the amount of about 115 mEq. per liter. Potassium is present in the extracellular fluid at the much lower level of 5 mEq. per liter. It has been previously emphasized that a cellular potassium deficit may or may not be accompanied by a decrease in the plasma potassium level below normal. In some instances of cellular potassium deficit, there may even be an elevated extracellular potassium.

The *balanced solutions* contain potassium in therapeutic amounts. This potassium is available to the body for the correction of both cellular and extracellular potassium deficits. It should be recalled that several days of treatment are necessary to correct cellular potassium deficits. This is true because any potassium entering the body must first traverse the extracellular fluid before reaching the cellular fluid. It must then pass through the walls of the cells.

The body homeostatic mechanisms attempt to prevent the extracellular fluid potassium from rising above a level of 5.5 mEq. per liter. It is, therefore, often necessary for the kidney to excrete much of the incoming potassium in spite of a cellular deficit in order to maintain the extracellular potassium level within the limits of normal. Thus, a prolonged period is required to repair the cellular potassium deficit.

Potassium Excess

The *balanced solutions* should not be used in the presence of a potassium excess. The treatment of this imbalance will be discussed in Chapter 28.

Calcium Deficit

The *balanced solutions* are not indicated for the treatment of calcium deficit. This imbalance will be discussed in Chapter 26.

Calcium Excess

The *balanced solutions* are not indicated for the treatment of calcium excess. This imbalance will be discussed in Chapter 28.

Base Bicarbonate Deficit (Metabolic Acidosis)

A base bicarbonate deficit results when the normal 1 to 20 ratio of carbonic acid:base bicarbonate is altered because of a decrease in the bicarbonate ions. The teeter-totter is tipped to the left. It has previously been noted that this decrease is secondary to the replacement of the bicarbonate by other anions, such as the chloride ion, organic acid anions or anions derived from ketone bodies such as acetoacetic acid and betahydroxybutyric acid.

In this imbalance the object of fluid therapy is to provide bicarbonate ions, to promote the excretion of the nonbicarbonate anions, and to overcome the processes responsible for the production of the ketone bodies.

Each of the parenteral *balanced solutions* described contains 20 or more mEq. of lactate ion in each liter of solution. This lactate is removed and metabolized in the liver. Bicarbonate ions, formed in the normal course of metabolism, replace the lactate ions quantitatively. As far as the clinical effect is concerned, these solutions provide 1 mEq. of bicarbonate ion for each 1 mEq. of lactate ion which they contain. The citrate ions in oral *balanced solution* will be metabolized and replaced by bicarbonate ions in the same manner as the lactate ions.

The patient with functional homeostatic mechanisms who suffers from a deficit of bicarbonate ions will be able to selectively retain lactate or citrate ions (or the bicarbonate ions that have replaced the lactate or citrate ions) for the relief of his base bicarbonate deficit.

The free water in the solution helps meet the body's need for water and helps the kidneys "wash out" nonbicarbonate anions.

The carbohydrate present in the solution will provide the body with a nonfat energy source. This decreases the need to metabolize body fat, a source of the ketone bodies which often play a significant part in the genesis of base bicarbonate deficit.

The occasional patient with a severe base bicarbonate deficit (metabolic acidosis), as manifested by a plasma bicarbonate below 2.5 mEq. per liter or a plasma pH below 7.15, requires initial treatment with sodium lactate or sodium bicarbonate solutions (see Chapter 26).

Base Bicarbonate Excess (Metabolic Alkalosis)

A base bicarbonate excess results when the normal 1 to 20 ratio of carbonic acid:base bicarbonate is altered because of an increase in the bicarbonate ions. The teeter-totter is tipped to the right. The object of fluid therapy is to provide nonbicarbonate anions, such as chloride ions, which can be selectively retained by the body homeostatic mechanisms in the place of the excess bicarbonate ions.

Since base bicarbonate excesses are almost invariably associated with some degree of potassium deficit, potassium should also be administered. Failure to provide potassium will almost always prevent a permanent correction of the base bicarbonate excess, particularly if parenteral fluid therapy is the only means available to correct the excess.

The *balanced solutions* contain adequate amounts of both chloride and potassium ions for therapy of base bicarbonate excess. The body homestatic mechanisms will selectively retain the chloride ion; the potassium ion is used to correct the potassium deficit that is practically always present.

The occasional patient with a severe base bicarbonate excess (metabolic alkalosis), as manifested by a plasma bicarbonate above 50 mEq. per liter, plasma chloride below 70 mEq. per liter or plasma pH above 7.65, may require initial treatment with ammonium chloride solutions (see Chapter 26).

Carbonic Acid Deficit (Respiratory Alkalosis)

A carbonic acid deficit results when the normal 1 to 20 ratio between carbonic acid and base bicarbonate is altered by a decrease in the carbonic acid. The teeter-totter is tipped to the right. The carbonic acid deficit occurs because of excessive losses of carbon dioxide through hyperventilation. *Correction of the cause of the hyperventilation is of primary importance in the correction of this imbalance.* If hyperventilation has been prolonged, an electrolyte concentration excess may occur because of excessive losses of water in the expired air.

Parenteral fluid therapy is of secondary importance. Its function is to support the compensatory efforts of the kidney, which attempts to correct this imbalance. The kidney attempts to produce a normal 1 to 20 ratio between carbonic acid and base bicarbonate by selectively retaining nonbicarbonate anions such as chloride ions and excreting bicarbonate ions. The object of parenteral fluid therapy is to provide chloride ions that can be used to replace bicarbonate ions, and free water that might be necessary to replace the water lost from the lungs because of hyperventilation.

The *balanced solutions* contain adequate amounts of both chloride and free water and thus will be useful as *supportive* therapy in this imbalance.

Carbonic Acid Excess (Respiratory Acidosis)

A carbonic acid excess results when the normal 1 to 20 ratio of carbonic acid:base bicarbonate is disturbed because of an increase of carbonic acid. The teeter-totter is tipped to the left. This increase of carbonic acid is secondary to retention of carbon dioxide because of hypoventilation. *The treatment of the cause of the hypoventilation is of primary importance in correction of this imbalance.*

As with carbonic acid deficits, parenteral fluids are of secondary importance. Their function is to lend support to the kidney which attempts to compensate for the carbonic acid excess by selectively retaining bicarbonate ions and rejecting nonbicarbonate anions. Fluid therapy is useful since either lactate, citrate or bicarbonate ions support the compensatory efforts of the kidney.

The parenteral *balanced solutions* all contain significant amounts of the lactate ion. The oral *balanced solution* contains significant amounts of the citrate ion. As explained previously, the provision of lactate or citrate ions is clinically equivalent to the provision of bicarbonate ions.

PREVENTIVE MAINTENANCE

Parenteral fluid therapy is useful not only for the treatment of specific fluid imbalances, but also for their *prevention*. Thus, an individual in normal fluid balance unable to take fluids orally must have his body fluids maintained parenterally.

The object of maintenance is to provide water and electrolytes in amounts that will meet normal requirements yet not be excessive. A parenteral solution that could provide these substances in the amounts normally ingested would be ideal. The *balanced solutions* are so formulated that they achieve this ideal, because when given in proper doses they simultaneously provide maintenance amounts of water and electrolytes. Ketosis and tissue breakdown of fasting, while not entirely eliminated, can be kept to a minimum by the carbohydrate incorporated in these solutions. *Balanced* *solutions* should not be employed for maintenance if serious impairment of the body homeostatic mechanisms is present.

SUMMARY

1) Clinical conditions involving one or more of the following changes in properties of extracellular fluid constitute the great majority of fluid imbalance states:

a) Volume deficit.
b) Electrolyte concentration excess.
c) Potassium deficit.
d) Base bicarbonate deficit.
e) Base bicarbonate excess.
f) Carbonic acid deficit.
g) Carbonic acid excess.

2) Fluid therapy for these imbalances can be provided by a simple regimen involving a *pump priming solution*, a *balanced solution* and a *gastrointestinal replacement solution*.

3) In therapy, the physician proceeds as follows:

a) If renal depression, as defined in Chapter 20, is present, he administers a *pump priming solution*. For dosage see Chapter 22.

b) After adequate renal function is established, he administers No. 75 *balanced solution* or No. 90 *balanced solution* to older infants, children or adults, or No. 48 *balanced solution* to full term and larger premature infants under 1 month of age in order to provide water and electrolytes for maintenance and to correct fluid volume deficits. Oral *balanced solution* can be substituted if oral administration is feasible. For dosage see Chapter 22.

c) Concurrent losses of gastric or intestinal secretions should be replaced by the suitable *gastrointestinal replacement solution*. Concurrent losses of perspiration or watery diarrheal stools should be replaced by a *balanced solution*. Estimated volume of concurrent losses are replaced by an equal volume of the appropriate replacement solution.

4) Volume of *pump priming solution* given is subtracted from the calculated dose of *balanced solution*. Solutions given for re-

placement of concurrent losses are additional to the volume of *pump priming solution* and *balanced solution* given.

5) *Balanced solutions* are also ideally suited for *prevention* of fluid imbalances in patients in normal fluid balance who are unable to take fluids by mouth.

6) The simple regime outlined above is not suitable for treatment of patients with the following changes in properties of extracellular fluid:

a) Fluid volume excess.
b) Electrolyte concentration deficit.
c) Potassium excess.
d) Calcium deficit.
e) Calcium excess.

Management of these numerically minor imbalances as well as of other special therapeutic problems will be covered in subsequent chapters.

24

Balanced Solutions: Their Limitations

Art is limitation; the essence of every picture is the frame.
GILBERT KEITH CHESTERTON

A RELATIVELY small number of patients with disturbances in the properties of extracellular fluid cannot be adequately or safely treated with the *balanced solutions.* Such patients can usually be recognized on the basis of a careful clinical history and physical examination, although confirmatory laboratory tests are frequently useful. The patients in whom the *balanced solutions* should not be used fall into four groups. In this chapter these four groups will be described in outline form to help the clinician gain a broad, over-all view of the limitations of the *balanced solutions.* Each group of patients will then be discussed in detail in chapters devoted specifically to them.

The first group includes those *patients who have severe dysfunction of one or more of the homeostatic mechanisms* responsible for the normalization of body fluids. These dysfunctions may in themselves give rise to specific fluid imbalances or their presence may preclude the use of the *balanced solutions* for the treatment of any fluid imbalance that may develop. The patients in this group include those with:

1) Renocardiovascular dysfunction.
2) Adrenal dysfunction.
3) Pituitary dysfunction.
4) Parathyroid dysfunction.

The second group includes those *patients who have urgent needs and who require immediate therapy.* Many of these patients have functional homeostatic mechanisms. Included in this group are those with:

1) Acute deficit in electrolyte concentration.
2) Acute calcium deficit.
3) Severe base bicarbonate deficit.
4) Severe base bicarbonate excess.
5) Acute deficit of whole blood.

The third group includes *severely burned patients,* who usually have urgent needs for specific replacement therapy. They may also develop renal dysfunction.

The fourth group is comprised of those *patients who have certain excesses* in their extracellular fluid. They include those with:

1) Extracellular fluid volume excess.
2) Potassium excess.
3) Calcium excess.

SUMMARY

1) A relatively small number of patients with changes in the properties of extracellular fluid cannot be adequately treated with the simplified plan of therapy presented in the preceding chapters. Such patients include those with:

a) Severe dysfunction of the homeostatic mechanisms.
b) Urgent needs which require immediate therapy.
c) Severe burns.
d) Certain excesses.

25

When Body Homeostasis is Impaired

Important principles may and must be flexible.
ABRAHAM LINCOLN

THIS CHAPTER will consider the principles of therapy of patients with fluid imbalance who are also suffering from impairment of one or more of the homeostatic mechanisms. Detailed plans of treatment will not be presented, since therapy for each patient must be planned to fit his exact needs. The clinical causes of impaired function of the homeostatic mechanisms were discussed in detail in Chapter 7.

RENAL DYSFUNCTION

The causes of impaired function of the renal homeostatic mechanisms include:

1) Failure of plasma to reach the kidneys in sufficient amounts.
2) Diseases of the kidney itself.
3) Immaturity of kidney function as seen in the smaller premature infant.
4) Factors sometimes active during the postoperative period including:
    antidiuretic action of narcotics such as morphine,
    renal sodium blockade due to the "alarm" or "stress" reaction.

Failure of Plasma to Reach Kidneys

Failure of plasma to reach the kidney in sufficient amounts may be secondary to heart failure, peripheral vascular collapse or acute deficit in blood volume. The kidneys may or may not be normal.

However, they cannot exert their controlling effect on extracellular fluid volume, electrolyte concentration or electrolyte composition unless they have adequate blood flow. The object of treatment is to restore normal heart action, re-establish normal vascular tone, or replace blood volume deficits. The *balanced solutions* cannot be used to achieve any of these therapeutic goals.

In heart failure a low sodium intake is indicated. Most physicians prefer to prescribe a sodium intake of less than 400 mg. per day in adults and proportionately less in infants and children. The *balanced solutions* contain more than this amount of sodium.

Since patients with heart failure may develop any fluid imbalance which occurs in patients with normal hearts, they may on occasion require relatively large amounts of sodium. A typical example is the patient who develops an electrolyte concentration deficit (low sodium syndrome) as the result of the combined use of a mercurial diuretic and a low sodium diet.

When parenteral fluid therapy is necessary in patients with heart failure, the physician should give only the minimal amounts of water and electrolytes necessary for maintenance or for correction of critical deficits. He must strike a balance between problems associated with heart failure and problems associated with fluid imbalances.

The potential hazard of administering solutions containing potassium to patients undergoing surgical procedures deserves emphasis. Renal depression caused by a fall in blood pressure during operation, may cause dangerous elevation of the plasma potassium if a solution containing this ion is being infused.

Some authorities administer potassium-containing solutions such as the *balanced solutions* during surgical operation provided the function of the body homeostatic mechanisms does not appear imperiled. Certainly if such infusions are given, they should be administered at a slow rate. Other authorities believe that potassium-containing solutions should never be given during surgical operations. Instead, they employ potassium-free solutions with composition similar to that of the *pump priming solutions.*

Diseases of the Kidney Itself

Intrinsic renal disease, regardless of the cause, interferes with the functional integrity of either the renal glomeruli, renal tubules or both. Although the kidney has considerable "reserve," there is a point in chronic kidney disease beyond which the kidney is no longer able successfully to control the water and electrolyte content of the extracellular fluid. Patients whose kidney disease has progressed beyond this critical point must be treated with solutions tailored to their exact needs, that is, solutions made up by the physician to meet as precisely as possible the patient's estimated need for water and electrolytes. This is necessary because the patient's homeostatic mechanisms are no longer capable of selectively retaining or rejecting these substances in accordance with body needs. Such therapy requires careful laboratory supervision combined with accurate calculations of the patient's water and electrolyte requirements. Weisberg in his textbook *Water, Electrolyte and Acid-base Balance* describes in detail the steps and calculations necessary to compound tailored solutions.

Acute renal insufficiency caused by renal tubular degeneration following shock, intravascular hemolysis, severe trauma or absorption of renal toxins, is potentially reversible. With proper management, many patients with this type of renal insufficiency will survive and will eventually have normal kidney function. Tailored solutions and careful laboratory supervision are required in the treatment of these patients. Hendricks in his recent article "The Conservative Management of Acute Renal Insufficiency" and Welt in his textbook *Clinical Disorders of Hydration and Acid-base Equilibrium* have described in detail a therapeutic regime for treatment of this type of kidney disease.

Immaturity of Kidney Function

Physiologic immaturity of renal function during the first few days of life of the smaller premature infant precludes the routine use of potassium-containing parenteral solutions such as No. 48 *balanced solution*. Unless renal function in such infants is known to be adequate, parenteral fluids used to meet their water and electrolyte requirements should be tailored to meet the individual infant's exact requirements as nearly as is possible.

Factors Active Postoperatively

Temporary dysfunction of the renal homeostatic control, manifested by excessive retention of water and sodium, sometimes occurs postoperatively. At least two factors can contribute to such partial failure of renal homeostasis. First, narcotics such as morphine, particularly if given in large doses, stimulate production of the antidiuretic hormone of the posterior pituitary gland. The excess hormone stimulates the cells of the kidney tubules to absorb increased amounts of water from the glomerular filtrate. Second, the stress of surgical operation frequently causes an increased production of adrenal cortical hormones, which, in turn, stimulate the cells of the kidney tubules to absorb abnormally great amounts of sodium from the glomerular filtrate.

A third factor, resulting paradoxically from *proper* functioning of the body homeostatic mechanisms, sometimes favors retention of water and sodium after operation. For example, the patient who has been subjected to preoperative fasting and who has lost substantial amounts of water and sodium chloride in perspiration during operation, is likely to enter the postoperative period with deficits of water and sodium. The body homeostatic mechanisms in such a patient act to conserve these substances.

In some few patients, all three factors may contribute to water and sodium retention. Routine administration of substantial amounts of either carbohydrate-in-water solution or isotonic solution of sodium chloride is contraindicated during the postoperative period. On the other hand, the use of *balanced solutions* during this phase at the maintenance dosage level of 1500 ml. per square meter of body surface per day, has not appeared to favor excessive retention of sodium and water even in patients with mild degrees of impairment of the body homeostatic mechanisms. Nevertheless, the physician should be alert to such retention especially when moderate or severe impairment of body homeostasis is present.

ADRENAL DYSFUNCTION

Patients with adrenal insufficiency tend to have a low plasma sodium, elevated plasma potassium and a decreased ability to excrete water. The *balanced solutions* with their relatively high potassium content, low sodium content and free water should not be given. Parenteral fluid therapy, which should be accompanied by careful laboratory supervision, is aimed at providing large amounts of sodium chloride. Generous quantities of carbohydrate may also be necessary to combat hypoglycemia. Appropriate amounts of necessary adrenocortical hormones must also be prescribed.

PITUITARY DYSFUNCTION

Diabetes insipidus is usually due to lack of production of the water-conserving (antidiuretic) hormone secondary to disease of the posterior pituitary gland. It can also result from failure of the kidney tubules to respond to the action of the water-conserving hormone. The major problem in this disease is the failure of the kidney to conserve water. The *balanced solutions* may be used to meet the electrolyte requirements of such patients, but the greater portion of their immense water requirement should be supplied by carbohydrate-in-water solutions.

PARATHYROID DYSFUNCTION

The parathyroid gland controls the calcium and phosphorus levels of the plasma. There appears to be a reciprocal relationship between these two electrolytes. As the level of one increases, the level of the other decreases. In hypoparathyroidism, the plasma calcium tends to be decreased and the plasma phosphorus increased. The *balanced solutions* described in this handbook contain phosphorus. Their administration to a patient with hypoparathyroidism may cause a further lowering of the plasma calcium and the development of hypocalcemic tetany. However, solutions similar to the *balanced solutions*, except for the absence of phosphorus, may be used in the same doses as have been outlined for the *balanced solutions* in Chapter 22.

SUMMARY

1) The *renal* homeostatic mechanisms may be impaired because of circulatory failure, acute deficits in blood volume, intrinsic renal disease, immaturity of kidney function or factors active postoperatively. When the kidney can no longer exercise control over the water and electrolyte content of extracellular fluid, the physician must tailor parenteral solutions to meet the exact requirements of the patient for water and electrolytes.

2) Proper therapy for patients with impairment of the *adrenal* homeostatic mechanisms involves the judicious use of sodium chloride solutions, carbohydrate and adrenocortical hormones combined with careful laboratory supervision.

3) Impairment of the *pituitary* homeostatic mechanisms, as manifested clinically in diabetes insipidus, calls for the use of *balanced solutions* to meet electrolyte needs, and of carbohydrate-in-water solutions, in addition, to meet the great water requirements.

4) Impairment of the *parathyroid* homeostatic mechanisms requires special solutions similar to the *balanced solutions* except for the absence of phosphorus.

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Urgent Needs

Diseases desperate grown
By desperate appliance are relieved,
Or not at all.
William Shakespeare

Patients suffering from severe disruptions in electrolyte concentration or composition of the extracellular fluid, as well as those who have sustained acute losses of whole blood, frequently require immediate specific replacement therapy. Although many of these patients have functional homeostatic mechanisms, the magnitude of the deficit is so great, or it has occurred so rapidly, that the *balanced solutions* will not provide adequate correctional therapy. They can usually be employed to advantage after the urgent need has been met.

EXTRACELLULAR FLUID CONCENTRATION CHANGES

Deficit in Total Electrolyte Concentration of Extracellular Fluid

In this deficit the ratio of water to electrolyte in the extracellular fluid is increased above the normal of 3.3 ml. of water to each milliequivalent of electrolyte. This may occur when water accumulates more rapidly than electrolytes in extracellular fluid or when electrolytes are lost more rapidly than water from the extracellular fluid. Whatever the cause of the electrolyte concentration deficit, a decrease in the effective osmotic pressure of the body fluids exists.

An electrolyte concentration deficit is invariably secondary to a relative decrease in the concentration of sodium and chloride ions because they constitute more than 80% of the electrolytes present in extracellular fluid. The clinical causes and characteristic findings of this deficit have been discussed in Chapter 11.

The object of therapy is to re-establish the normal water-to-electrolyte ratio in the extracellular fluid. This is accomplished by the administration of sodium chloride solutions including:

1) Isotonic solution of sodium chloride, containing 154 mEq. of sodium and 154 mEq. of chloride per liter,

2) 3% sodium chloride solution, containing 513 mEq. of sodium and 513 mEq. of chloride per liter, or

3) 5% sodium chloride solution, containing 862 mEq. of sodium and 862 mEq. of chloride per liter.

Ideally, treatment of the individual patient should be guided by the plasma sodium level. If a plasma sodium level is not obtainable, the sum of the plasma bicarbonate in milliequivalents and the plasma chloride in milliequivalents plus 12 mEq. can be substituted, unless there is an abnormal accumulation of other anions which may displace bicarbonate or chloride. The significance of the plasma sodium level as an indication for parenteral therapy, depends heavily upon the clinical status of the patient, with particular reference to how the deficit developed. This subject was discussed in detail in Chapter 11. If no laboratory determinations are available, a plasma sodium of 130 mEq. per liter may be assumed in the presence of diagnostic clinical findings. Treatment in this instance must be guided entirely by the clinical response of the patient.

The total sodium deficit to be replaced can be roughly calculated as follows:

1) Subtract the patient's plasma sodium level in milliequivalents from the average sodium level of 142 mEq. per liter to obtain the *sodium deficit per liter.*

2) Multiply the patient's body weight in kilograms (pounds divided by 2.2) by the per cent of the body weight that is extracellular fluid (15% in children and adults and 25% in infants). This figure represents the patient's extracellular fluid weight in kilograms or his extracellular fluid volume in liters, since one liter of fluid weighs about one kilogram.

3) Multiply the *sodium deficit per liter* by the *extracellular fluid volume in liters.* This gives the *total sodium deficit in milliequivalents.* Correcting the sodium deficit with a suitable solution of sodium chloride automatically corrects the electrolyte concentration deficit.

In the presence of a *normal* extracellular fluid volume or an extracellular fluid volume *excess,* a 3% or 5% solution of sodium chloride should be used to correct the electrolyte concentration deficit. Only one-half of the calculated deficit should be corrected initially. Further therapy should await re-evaluation of the patient's clinical status and redetermination of his plasma sodium.

Hypertonic sodium chloride solutions should be administered intravenously at the rate of 1 ml. per square meter of body surface per minute.

A 70 Kg. man with a body surface of 1.7 square meters is suffering from an electrolyte concentration deficit. His plasma sodium level is 125 mEq. per liter and his extracellular fluid volume is normal. How much of a 3% sodium chloride solution should he be given? The calculation is as follows:

1) The patient's sodium deficit per liter of extracellular fluid is 142 mEq. minus 125 mEq., or 17 mEq. per liter.

2) His approximate extracellular fluid volume is 70 Kg. multiplied by 15%, or 10.5 liters.

3) The total sodium deficit in the extracellular fluid is 10.5 multiplied by 17 mEq., or 178 mEq. of sodium.

4) The volume of solution required to repair the deficit will be the fraction 178/513 of 1000 ml. The calculation is as follows:

$$\frac{178 \text{ x } 1000 \text{ ml.}}{513} = 350 \text{ ml.}$$

5) Initially, only one-half or 175 ml. of the solution should be given at the rate of 1.7 ml. per minute (1 ml. x 1.7). Further therapy should be guided by a redetermination of the plasma sodium level and reassessment of the clinical condition of the patient.

In the presence of an extracellular fluid volume *deficit,* an isotonic solution of sodium chloride should be used to correct the calculated electrolyte concentration deficit. The rate of administration for isotonic solution of sodium chloride is the same as for *balanced solutions.*

Patients who have normal homeostatic mechanisms can be treated with the *balanced solutions* after the concentration deficit has been corrected.

EXTRACELLULAR FLUID COMPOSITION CHANGES

Calcium Deficit

Tetany associated with a decrease in the amount of ionized calcium in the plasma often requires immediate replacement therapy. Calcium deficits not associated with tetany cannot be corrected by parenteral fluid therapy.

A 10% calcium gluconate solution is used most commonly for the immediate treatment of tetanic convulsions associated with hypocalcemia. The usual ampule size of the calcium gluconate solution is 10 ml. It should be administered intravenously. Each 10 ml. of a 10% solution of calcium gluconate contains 4.5 mEq. of calcium.

The recommended dose in children is 1 ml. of the 10% solution per kilogram of body weight, diluted with two volumes of isotonic solution of sodium chloride and given over a 10-minute period. The total dose in any one infusion should not exceed 10 ml. of the 10% calcium gluconate solution. In larger children and adults a dose of 10 ml. of the 10% calcium gluconate solution diluted with two volumes of isotonic solution of sodium chloride can be given. A decrease in heart rate below 60 per minute indicates that the rate of administration of the calcium gluconate solution is too rapid.

If the disease process which originally caused the hypocalcemia cannot be immediately controlled, it may be necessary to repeat the administration of the calcium gluconate. The physician should be guided by the clinical response of the patient.

Base Bicarbonate Deficit (Metabolic Acidosis)

A patient is seen occasionally with a severe base bicarbonate deficit (metabolic acidosis), as indicated by a plasma bicarbonate below 2.5 mEq. per liter or a plasma pH below 7.15. A sixth-molar sodium lactate solution or a sixth-molar sodium bicarbonate solution can be used as the initial treatment of such patients. These solutions contain 167 mEq. of sodium and 167 mEq. of either lactate or bicarbonate per liter. As previously noted, the lactate ion in the sodium lactate solution will be metabolized in the liver and replaced by bicarbonate ion.

Theoretically, administration of either of these solutions in doses of 1.8 ml. per kilogram of body weight for infants and young children or 0.9 ml. per kilogram of body weight for older children and adults will raise the plasma bicarbonate level about 1 mEq. per liter.

The physician should not attempt to raise the level of the plasma bicarbonate with these solutions more than 15 mEq. per liter in one infusion. Thus, he should not administer more than 27 ml. (15 x 1.8 ml.) per kilogram of body weight of either of these solutions to infants or young children. Nor should he administer more than 13.5 ml. (15 x 0.9 ml.) per kilogram of body weight of either of these solutions to older children or adults. The rate of administration of either of these solutions should not exceed the rates indicated for *balanced solutions*.

If the patient has functional homeostatic mechanisms, the *balanced solutions* can be used for further correction of the base bicarbonate deficit. If the patient does not have functional homeostatic mechanisms, careful laboratory supervision combined with use of tailored solutions is necessary.

Base Bicarbonate Excess (Metabolic Alkalosis)

Occasionally, a patient is seen with a severe base bicarbonate excess, as indicated by a plasma pH above 7.65, a plasma bicarbonate over 50 mEq. per liter, or a plasma chloride below 70 mEq. per liter. A sixth-molar solution of ammonium chloride can be used in the initial treatment of these patients. However, such treatment is hazardous and should be employed only in severe alkalosis.

Molar solutions of ammonium chloride are available in 50-ml. ampules. A sixth-molar solution can be prepared by adding two of the 50-ml. ampules of the molar solution to 500 ml. of a carbohydrate-in-water solution. The sixth-molar solution contains 167 mEq. of ammonium and 167 mEq. of chloride ion per liter.

Ammonium chloride solutions exert their effect in the following fashion:

1) The ammonium ions are rapidly metabolized in the liver.
2) The chloride ions released replace the bicarbonate ions in the extracellular fluid.

Theoretically, administration of a sixth-molar ammonium chloride solution in doses of 1.8 ml. per kilogram of body weight for infants and young children or 0.9 ml. per kilogram of body weight for older children and adults will raise the plasma chloride 1 mEq. per liter. The physician should not attempt to raise the level of the plasma chloride with these solutions more than 15 mEq. per liter. Thus, he should not administer more than 27 ml. (15 x 1.8 ml.) per kilogram of body weight to infants and young children. Nor should he administer more than 13.5 ml. (15 x 0.9 ml.) per kilogram of body weight to older children or adults. The rate of administration of this solution should not exceed 2 ml. per square meter of body surface per minute.

Patients receiving ammonium chloride solutions should be observed carefully from the clinical and the laboratory standpoints for evidence of ammonium ion intoxication or of overcorrection of the base bicarbonate excess. Further therapy should be confined to the use of the *balanced solutions* provided the patient has functional homeostatic mechanisms. These will provide needed potassium. Recall that a base bicarbonate excess is accompanied by a cellular potassium deficit; the ammonium chloride solution provides no potassium.

If the homeostatic mechanisms are not functional, solutions tailored to the patient's exact needs should be given.

WHOLE BLOOD DEFICIT

Approximately 10% of the body weight is whole blood, of which about 40% is comprised of cellular elements and 60% of plasma. The body of the average adult contains six or seven liters of whole blood. A severe deficit of either whole blood or plasma is manifested by shock. The primary cause of the shock is a decrease in the intravascular fluid volume. Secondarily, loss of blood vessel tone and heart failure may occur.

The object of therapy, particularly in the presence of shock, is the restoration of the intravascular fluid volume to normal. If the deficit is due to a loss of whole blood, it should be corrected with whole blood. Plasma or dextran can be used if whole blood is not immediately available. If the deficit is due to a loss of plasma, it should be corrected with plasma. Dextran can also be used if plasma is not available. Dextran should always be considered a temporary expedient and not a substitute for whole blood or plasma.

If the patient is in shock, the amount of blood, plasma or dextran to be used and the rate of its administration depend upon the clinical response of the patient as measured by pulse and blood pressure. If the patient is not in shock, the usual amount of any of these substances given at one time is 320 ml. per square meter of body surface over a period of three to four hours.

SUMMARY

1) Patients with severe deficits in electrolyte concentration or electrolyte composition of extracellular fluid and patients who have lost large amounts of blood require immediate specific replacement therapy.

2) A deficit in total electrolyte concentration of extracellular fluid can be gauged by the plasma sodium level. The *total sodium deficit* of the extracellular fluid can be calculated from the *sodium deficit per liter* and the *patient's weight in kilograms.*

3) If the extracellular fluid volume is *normal* or *excessive,* a 3% or 5% solution of sodium chloride should be administered. If an extracellular fluid volume deficit is *present,* an isotonic solution of sodium chloride should be used.

4) Convulsions associated with hypocalcemia can be treated with a 10% solution of calcium gluconate.

5) In a severe base bicarbonate deficit (metabolic acidosis) with the plasma pH below 7.15 or plasma bicarbonate below 2.5 mEq. per liter, either a sixth-molar sodium lactate or a sixth-molar sodium bicarbonate solution is used for initial therapy.

6) In a severe base bicarbonate excess (metabolic alkalosis) with the plasma pH above 7.65, plasma bicarbonate over 50 mEq. per liter, or plasma chloride below 70 mEq. per liter, a sixth-molar solution of ammonium chloride can be used for initial therapy. Use of this solution is relatively hazardous.

7) A severe deficit of whole blood or plasma is manifested by shock, primarily caused by decreased plasma volume. If the deficit is caused by *whole blood* loss, it should be corrected by *whole blood*. Plasma or dextran can be used in emergencies. If the deficit is caused by *plasma* loss, it should be corrected with *plasma* or dextran if plasma is not available.

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The Patient is Burned

Double, double toil and trouble;
Fire burn and cauldron bubble.
William Shakespeare

Planned, intelligent fluid therapy is of crucial importance during the first several days following a severe burn. It may well decide the issue between rapid recovery and prolonged convalescence, between life and death. To provide such therapy the physician must first be aware of the dangers that threaten the patient's life during this critical period. The burned patient's ominous "four horsemen" of disaster include:

1) Losses of body fluids.
2) Shock.
3) Renal depression.
4) Remobilization of edema fluid.

These hazardous aftermaths of a burn are closely interrelated, as will be clear from the following discussion.

FOUR HORSEMEN OF DISASTER

Losses of Body Fluids

Both plasma and interstitial fluid are lost during a burn. Blood, a suspension of red cells in plasma, is also lost. Fluid losses occur in several ways.

First, a plasma-to-interstitial fluid shift occurs in the region of the burn. The magnitude of this shift depends upon the *area* of the burn and its *severity*. Clinically, the shift results in edema. During the first day or two in burns of more than a quarter of the body surface, the volume of the edema fluid may equal one-half of the total volume of the extracellular fluid. More than this volume of edema fluid does not usually accumulate regardless of the area of the burn. If it does, the patient rarely survives.

Consider a specific example: An adult with a body surface of 1.75 square meters has about 10.5 liters of extracellular fluid. Should he sustain a 50% burn, the volume of edema fluid formed during the first day or two would approximate 5.25 liters. This quantity exceeds the total plasma volume of the patient. Obviously, all of the edema fluid is not derived from the plasma; some of it comes from the body cells and some from administered fluids.

Second, fluid is lost through "leakage" of about equal amounts of plasma and interstitial fluid from the burned surface. As would be expected, this fluid contains half the amount of protein normally present in plasma. The amount of fluid lost in this manner is proportional to the skin area burned in burns up to 50% of the skin area. Such losses do not increase appreciably in burns of more than 50%.

A third source of fluid loss is leakage of blood from the damaged capillaries and small blood vessels of the burned surface. Some blood may be destroyed at the time of the burn. The amount of blood lost during the first two days rarely exceeds 10 to 15 ml. per kilogram of body weight.

Finally, the burned patient has losses of fluid not related to the burn. Water lost as insensible perspiration and urine equals about 1500 ml. per square meter of body surface per day.

Shock

The danger of shock hovers over the badly burned patient during the critical first two or three days. The causes of shock include the decrease in plasma volume, pain and fear. Its clinical picture of pallor, apprehension, falling blood pressure and rising pulse rate is all too familiar to the physician.

Renal Depression

Renal depression frequently occurs in severe burns as the result of the decrease in plasma volume, of previous disorders such as chronic heart disease or chronic nephritis, or of kidney damage from such effects of the burn as anoxia and hemoglobinuria.

Unless the depression of kidney function is corrected, it will lead to such imbalances as potassium excess, extracellular fluid volume excess, base bicarbonate deficit (metabolic acidosis) and the accumulation of metabolic waste products such as urea. If the kidney is damaged to the extent that its homeostatic functions are seriously impaired, the problems of therapy are usually insurmountable.

Remobilization of Edema Fluid

Remobilization of edema fluid represents an interstitial fluid-to-plasma shift which begins on the second or third day after the patient has been burned. Its usual duration is from 24 to 72 hours. Recall that the volume of edema fluid in a burn may equal the total normal plasma volume. Fatal acute pulmonary edema may occur because the renocardiovascular system is not capable of handling the volume of water and electrolytes shifting from the interstitial fluid into the plasma.

FLUID THERAPY FOR THE BURNED PATIENT

Area of Skin Burned

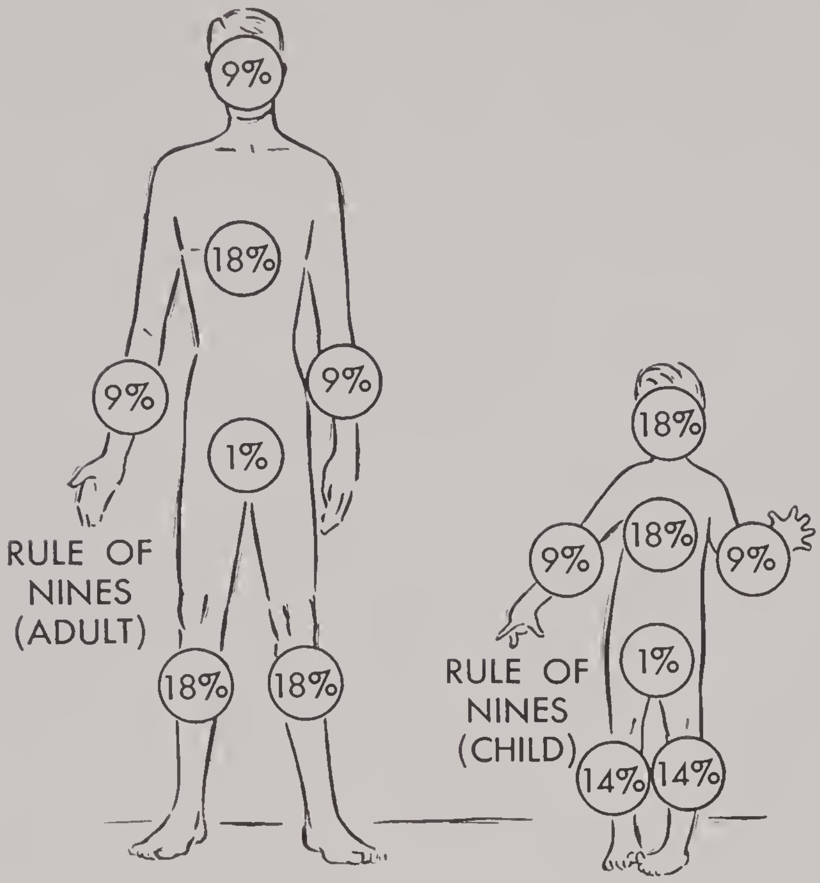
In order to determine the approximate total fluid need of the burned patient, it is first necessary to estimate the area of skin that has received *second* or *third degree burns.* The "rule of nines" is a convenient help. Its use is based on the following tabulation showing body parts and the approximate per cent of body surface contributed by the skin covering the part:

1) Head, 9%.
2) Each arm, 9%.
3) Front of trunk, 18%.
4) Back of trunk, 18%.
5) Each leg, 18%.
6) Genitalia, 1%.

The above values are for older children and adults.

The following approximate values apply to infants and young children, with their larger heads and shorter legs:

1) Head, 18%.
2) Each arm, 9%.



3) Front of trunk, 18%.
4) Back of trunk, 18%.
5) Each leg, 14%.
6) Genitalia, 1%.

In calculating the per cent of skin area burned, one simply adds up the per cent figures for the burned parts. For example, an adult with second or third degree burns of one arm (9%), the front of the trunk (18%) and half of one leg (9%) would have 36% of his skin area burned, or a 36% burn.

Blood Required for Therapy

Not more than 10 to 15 ml. of whole blood per kilogram body weight is needed during the first few days after a severe burn. It is given late on the first day or during the second day. To give more is dangerous, since there has been a greater loss of plasma than of red cells from the vascular system. If an excessive quantity of blood is given, the hematocrit will rise, and the viscosity of the blood may increase to the point where the circulation is imperiled.

Take as an example a 70 Kg. (154 lb.) adult who has been burned: 10 ml. of blood per kilogram body weight x 70 equals 700 ml., the amount of blood required. We will assume it is given late on the first day, about 16 hours after the burn.

Plasma Required for Therapy

Talbot and associates suggest that on the first day 60 ml. of plasma per square meter of body surface be given for each 1% of the skin which has received a second or third degree burn. Our 70 Kg. adult had a surface area of 1.75 square meters. He sustained a 36% burn. Multiplying 60 ml. x 1.75 x 36 we arrive at the figure of 3780 ml. as the volume of plasma indicated.

On the day whole blood is given, its amount is subtracted from the calculated volume of plasma to be administered for that day. Since 700 ml. of whole blood was given to our mythical patient on the first day, this amount should be subtracted from the volume of plasma calculated. Subtracting 700 ml. from 3780 ml. leaves 3080 ml. or about three liters of plasma to be given on the first day.

The amount of plasma given on the second day should ordinarily not be greater than 30 ml. per square meter of body surface for each 1% of the skin which has received a second or third degree burn. Our patient with a body surface of 1.75 square meters would receive 30 ml. x 1.75 x 36 or 1890 ml., roughly 1900 ml. of plasma.

After the second day plasma is usually not necessary.

Electrolyte Solution

As Talbot and associates have suggested, a solution somewhat similar in electrolyte concentration and composition to interstitial fluid, except for the absence of potassium, should be administered to replace loss of interstitial fluid from the surface of the burn. This solution should not contain potassium for two reasons:

1) As a result of the destruction of tissue that occurs following an extensive burn, the extracellular fluid is likely to be flooded with potassium.

2) Renal function is frequently impaired following burns. Potassium should never be administered parenterally when such a possibility exists.

A fairly suitable electrolyte solution for replacement of lost interstitial fluid is isotonic solution of sodium chloride. Such a solution contains in each liter:

sodium	154 mEq.
chloride	154 mEq.

A solution having the advantage that its chloride content approximates that of extracellular fluid is "saline lactate," devised by Talbot and his group. It contains in each liter:

sodium	140 mEq.
chloride	110 mEq.
lactate	30 mEq.

On the first day one of these solutions should be administered to the burned patient in the amount of 20 ml. per square meter of body surface for each 1% of skin which has received a second or third degree burn. Our patient, with his 1.75 square meters of body surface, would receive 20 ml. x 1.75 x 36, which equals 1260 ml., roughly 1200 ml. of the electrolyte solution.

The amount of electrolyte solution given on the second day should not ordinarily exceed 10 ml. per square meter of body surface for each 1% of skin which has received a second or third degree burn. Thus our patient with a body surface of 1.75 square meters would receive 10 ml. x 1.75 x 36, which equals 630 ml., roughly 600 ml., on the second day.

It is usually not necessary to administer the electrolyte solution after the second day.

Carbohydrate-in-Water Solution

Burned patients need water unaccompanied by electrolyte to replace water losses from the skin and lungs and in the urine. Talbot and associates suggest a dosage level of 1500 ml. per square meter of body surface per day for the first two post-burn days.

Our patient with a body surface of 1.75 square meters would receive 1500 ml. x 1.75 or 2625 ml., roughly 2500 ml. of a carbohydrate-in-water solution on both the first and second day after the burn.

Balanced Solution

On the third day and on each day thereafter while the patient is on a parenteral fluid regime, he should be given a *balanced solution*, 1500 ml. per square meter of body surface, provided:

1) There is no evidence of impairment of the homeostatic mechanisms.

2) There is no danger of overloading the renocardiovascular system because of an interstitial fluid-to-plasma shift secondary to remobilization of edema fluid.

No. 75 *balanced solution* or No. 90 *balanced solution* should be given to an adult or child; No. 48 *balanced solution* is proper for an infant under one month of age.

Our patient with a body surface area of 1.75 square meters would receive 1500 ml. x 1.75 which equals 2625 ml., roughly 2500 ml. of a *balanced solution* per day.

Need for Caution

If there is clinical or laboratory evidence of an interstitial fluid-to-plasma shift caused by remobilization of edema fluid, the rate of administration of parenteral fluids should be reduced or administration temporarily discontinued depending upon the rapidity of the shift. Such shifts usually occur on the second or third day, although they may occur later. They are made manifest by the clinical and laboratory findings described in Chapter 12.

Summary of First Day's Fluid Therapy

Our 70 Kg. adult, with a body surface of 1.75 square meters, and a 36% second or third degree burn would thus receive a total of 7400 ml. of fluids during the first 24 hours following his burn. This would include:

1) 700 ml. of whole blood.
2) 3000 ml. of plasma.
3) 1200 ml. of isotonic solution of sodium chloride.
4) 2500 ml. of a carbohydrate-in-water solution.

Summary of Second Day's Fluid Therapy

Our hypothetical patient would receive a total of 5000 ml. of fluids on the second day following the burn. This would include:

1) 1900 ml. of plasma.
2) 600 ml. of isotonic solution of sodium chloride.
3) 2500 ml. of a carbohydrate-in-water solution.

Fluid Therapy After the Second Day

If our patient required intravenous fluid therapy after the second day, a *balanced solution* would be administered, provided there was no impairment of the homeostatic mechanisms and no danger of overloading the renocardiovascular system. Such therapy might include 2500 ml. of No. 75 *balanced solution,* the maintenance quantity of *balanced solution.*

Rate and Route of Administration

Fluids are usually administered intravenously to burned patients. If the patient can tolerate fluids by mouth, this route may be used for the carbohydrate-in-water solution, for the isotonic solution of sodium chloride or saline lactate, and for the *balanced solution.*

One-half of the calculated first day's fluid should be given during the first eight hours. The other half should be administered in the following 16 hours. On the second day, the entire 24-hour period should be utilized for fluid administration in view of the hazards of the remobilization of edema fluid that starts near the end of the second day.

The rate of fluid administration may be increased or decreased at any time in accordance with the progress of the initial plasma-to-interstitial fluid shift or the secondary interstitial fluid-to-plasma shift. The progress of these shifts can be followed by serial determinations of hemoglobin or hematocrit and by urinary flow.

Either the hematocrit or hemoglobin and the rate of urinary excretion should be determined every hour duing the first 48 hours. The hematocrit should be maintained between a level of 40 and 50%, and the hemoglobin between 11 and 15 Gm. per 100 ml. A retention catheter should be placed in the patient's bladder to facilitate measurement of urinary flow, which should be maintained at a rate of between 10 and 30 ml. per square meter of body surface per hour. For the patient with a surface area of 1.75 square meters, this represents a flow of between 18 and 53 ml. per hour which may be difficult to maintain during the first few hours when there is a deficit in plasma volume plus shock or impending shock. A much greater urinary flow may occur during the remobilization of edema fluid.

GENERAL THERAPY

While one is attending to the acute fluid balance problems of the burned patient, other needs should be kept in mind. Pain and fear must be treated. Narcotics should be avoided if at all possible; sedation by means of barbiturates usually suffices. Reassurance and attention by the physician go far toward improving the mental state of the patient.

Antibiotics and other anti-infective agents are indicated to prevent or minimize infection. Appropriate measures for prevention of tetanus should be carried out. Careful attention to the provision of an adequate airway may be life-saving.

THERAPY FOLLOWING THE CRITICAL PERIOD

If the patient survives the critical period, his needs are somewhat changed. As Blocker, Pareira, and Mindrum have all emphasized, good nutrition is of *first importance.* Burned patients have great nutritional needs, several times those of the healthy individual. Frequent oral feedings of foods or commercial mixtures high in protein, calories and vitamins should be started as soon as possible. If the patient refuses oral feedings, tube feedings may be employed.

Extremely well tolerated, high protein tube feeding mixtures are commercially available and can be given by polyvinyl nasogastric tube. Polyvinyl tubing can be left in place for days with minimal discomfort. Such tubing is greatly superior to the old bulky rubber tubing of a few years ago. Vitamin preparations containing at least members of the B complex and vitamin C should be administered.

Skin grafting should be started as early as possible, in general, when a healthy bed of granulation tissue is present. Successful skin grafting is in large measure dependent upon adequate nutrition.

While providing the burned patient with optimal nutrition pays rich dividends, failure to meet his nutritional needs may lead to what Blocker terms "burn decompensation." This state is characterized by weight loss, decreased resistance to infection, anorexia, failure of skin grafts to take, cachexia and death.

Early ambulation, even in severely burned patients, improves appetite, helps to prevent contractures and sustains the patient's morale.

Certainly, double toil and double trouble characterize the clinical problem of the severely burned or scalded patient. Skill, care and devotion in rich measure are required of the physicians and nurses in whose hands the destinies of such patients so largely lie.

SUMMARY

1) The hazardous aftermaths of a burn include:
    a) Losses of body fluids including plasma, interstitial fluid and whole blood.
    b) Shock.
    c) Renal depression.
    d) Remobilization of edema fluid.

2) It is necessary to estimate the area of skin which has received a second or third degree burn to arrive at a proper dosage. The "rule of nines" is useful for this estimate.

3) From 10 to 15 ml. of whole blood per kilogram of body weight may be given on the first or second day following a severe burn.

4) 60 ml. of plasma per square meter of body surface is given on the first day for each 1% of the skin which has received a second or third degree burn. One-half this amount is given on the second day. On the day whole blood is given, its amount is subtracted from the volume of plasma to be administered.

5) An electrolyte solution such as isotonic solution of sodium chloride should be administered to the burned patient on the first day in the amount of 20 ml. per square meter of body surface for each 1% of skin area which has received a second or third degree burn. One-half this amount of the electrolyte solution should be given on the second day.

6) A 5 or 10% carbohydrate-in-water solution, 1500 ml. per square meter of body surface, should be given on the first day. The same amount should be given on the second day.

7) If there is evidence of an interstitial fluid-to-plasma shift caused by remobilization of edema fluid on the second or third day, parenteral fluid therapy should be curtailed or temporarily discontinued.

8) A *balanced solution,* 1500 ml. per square meter of body surface, should be given on the third day and on each day thereafter while the patient is receiving parenteral fluids.

9) While fluids are usually administered intravenously to severely burned patients in the acute phase of the burn, the oral or nasogastric route may prove useful, particularly after the second day.

10) Hematocrit, hemoglobin and urinary flow rate are useful gauges for regulation of therapy.

11) Optimal nutrition speeds the recovery of the burned patient.

12) The management of a severely burned patient requires a skilled therapeutic team and constant laboratory control.

28

Certain Excesses

> *The first water cure was the Flood, and it killed more than it cured.*
>
> Charles Lamb

Among the five changes in properties of extracellular fluid that cannot be managed by the simplified system of therapy presented in Chapters 17 to 23 are three excesses:

1) Extracellular fluid volume excess.
2) Potassium excess.
3) Calcium excess.

Treatment of each is aimed at the *removal* of a single electrolyte or of a combination of water and electrolytes from the extracellular fluid. This is in contrast to management of the other changes in properties of extracellular fluid whose treatment has as its goal the *addition* of water and electrolytes. Let us consider the management of these three excesses.

EXTRACELLULAR FLUID VOLUME EXCESS

The object of treatment of this imbalance is to reduce the volume of the extracellular fluid without producing abnormal changes in its electrolyte concentration or composition. Treatment varies with the cause of the imbalance and with the functional status of the patient's homeostatic mechanisms.

If the cause is excessive administration of a parenteral fluid such as isotonic solution of sodium chloride, simple discontinuance of such infusions may be adequate in patients having functional homeostatic mechanisms. Administration of a *balanced solution* will provide free water to aid in the excretion of excess electrolytes while, at the same time, meeting the electrolyte requirements of the patient for potassium and other ions which may be in deficit. Ordinarily, the maintenance dose of 1500 ml. per square meter of body surface of the appropriate *balanced solution* should be employed. Patients not having functional homeostatic mechanisms may require mercurial diuretics or phlebotomy.

If the volume excess has developed over a period of time, withholding of fluids is not indicated. This type of volume excess may occur in congestive heart failure, chronic kidney disease or chronic liver disease with portal hypertension. The homeostatic mechanisms are not functioning normally in patients with these disorders. Treatment of the underlying disease process often aids in the relief of the latter type volume excess. Specific treatment of the excess includes the use of a low sodium diet and various types of diuretic agents. Occasionally, a more radical procedure, such as phlebotomy, abdominal paracentesis or thoracentesis, must be performed.

Patients with chronic malnutrition may develop a volume excess because of a plasma protein deficit and other factors. The volume excess that develops in congestive heart failure, chronic renal disease or chronic liver disease is often due in part to malnutrition. Treatment in these instances is aimed at improving the nutritional status of the patient.

POTASSIUM EXCESS

Potassium excess is usually secondary to a temporary or chronic dysfunction of the body homeostatic mechanisms. Occasionally, it may be secondary to overzealous parenteral administration of solutions containing potassium.

The object of therapy is to remove the excess potassium from the extracellular fluid. The method used will depend upon the status of the patient's homeostatic mechanisms, and, in some instances, on the availability of suitable equipment and trained personnel. Currently employed methods include:

1) Elimination of potassium intake by use of a potassium-free diet or potassium-free solution.

2) Oral or intravenous administration of large amounts of sodium chloride.

3) Use of a combination of insulin and dextrose.
4) Administration of testosterone.
5) Administration of desoxycorticosterone acetate.
6) Extrarenal dialysis.
7) Administration of carbonic anhydrase inhibitors.
8) Administration of ion exchange resins.
9) Intravenous administration of calcium.

These methods of treatment all require careful laboratory supervision.

Diminished Potassium Intake

The use of a low potassium or potassium-free diet is indicated in individuals whose homeostatic mechanisms are not functioning properly. Examples of this type of patient include those with chronic kidney disease or adrenal insufficiency.

Potassium-free fluids should be used in individuals whose kidney function is temporarily depressed. *Pump priming solutions* are potassium-free solutions. Patients with chronic kidney or adrenal disease should not receive potassium-containing solutions unless there is specific evidence of a potassium deficit. In this latter case potassium can be given, but with caution.

Increased Sodium Chloride Intake

The use of large amounts of sodium chloride parenterally or orally combined with a potassium-free fluid or diet is helpful in the treatment of potassium excess in patients whose kidneys are functional. There appears to be a reciprocal relationship between sodium and potassium; an intake of large amounts of sodium results in an increased loss of potassium in the urine.

Insulin Plus Dextrose

The use of intravenous insulin and dextrose is the most satisfactory method of producing a temporary fall in the potassium level of the extracellular fluid. Insulin accentuates transformation of dextrose to glycogen. This glycogen along with extracellular potassium enters the cells thus reducing the level of potassium in the extracellular fluid. The measure is particularly useful in those patients whose homeostatic mechanisms are impaired.

A 25% dextrose solution containing one unit of insulin for each 2 Gm. of dextrose is employed. The dose of such a solution is 2 ml. per kilogram (2.2 pounds) of body weight. It can be repeated according to the need as indicated by repeated determinations of the plasma potassium level and serial electrocardiographic tracings.

Testosterone

Testosterone, the male sex hormone, stimulates the synthesis of new protein tissue. Potassium, the chief cellular cation, is an integral part of such tissue. Because of this relationship, daily intramuscular injections of 10 to 25 mg. of testosterone may be helpful in reducing plasma potassium levels in patients whose homeostatic mechanisms are not functioning normally. The action of testosterone is much slower than that of insulin and dextrose. Its effect is also temporary.

Desoxycorticosterone Acetate

Desoxycorticosterone acetate (DOCA) is a synthetic adrenocortical steroid. One of its major actions is to cause sodium retention by the kidneys. Secondarily, it causes a loss of potassium through the same organs. For many years, DOCA was the chief therapeutic agent for the adrenal insufficiency of Addison's disease. It may occasionally be useful for the treatment of potassium excess in patients not having Addison's disease, provided the kidneys are functioning. It should be used only under careful laboratory supervision. The dose varies from patient to patient and may be as little as 0.25 mg. or as much as 5 or 6 mg. per day.

Extrarenal Dialysis

Extrarenal hemodialysis consists of dialyzing the patient's blood against carefully formulated solutions by means of some type of artificial kidney. While this method is satisfactory for temporary treatment of a potassium excess, it is not generally available and requires a highly skilled therapeutic team and special equipment.

Other means of extrarenal dialysis include continuous or intermittent peritoneal or gastrointestinal lavage. These methods are not only complicated but also require skilled therapeutic teams. They are not as satisfactory as the use of an artificial kidney.

Carbonic Anhydrase Inhibitors

Recently, a group of drugs has been developed which inhibits the enzyme carbonic anhydrase. This enzyme accelerates chemical reactions which cause potassium in the glomerular filtrate to be exchanged for hydrogen in the cells of the kidney tubules. The carbonic anhydrase inhibitors diminish the activity of carbonic anhydrase and thus decrease the normal transfer of potassium from the filtrate into the cells of the kidney tubules. The result is an increase in urinary excretion of potassium. These inhibitors have the undesirable side effect of producing a base bicarbonate deficit (metabolic acidosis). The inhibitor most often used is acetazoleamide or Diamox®. Its oral dose is 5 mg. per kilogram (2.2 pounds) once a day or every other day.

Ion Exchange Resins

A specific ion exchange resin can be used orally or rectally for the purpose of promoting the fecal excretion of potassium. A resin "in the ammonium or hydrogen cycle," containing no potassium, is employed. Such a resin will exchange ammonium and hydrogen ions for sodium and potassium ions in the intestinal secretions. Considerable amounts of potassium can be removed in this way.

This resin has the disadvantage of producing a base bicarbonate deficit (metabolic acidosis) because of intestinal absorption of exchanged hydrogen and ammonium ions, combined with loss of potassium and sodium ions in the stools. It should not be used unless careful laboratory supervision is available. The base bicarbonate deficit (metabolic acidosis) should be treated by carefully calculated amounts of a sixth-molar sodium lactate or sodium bicarbonate solution. The average adult dose is 50 Gm. of the resin per day until the potassium level is normal.

Calcium Administration

In the latter part of the nineteenth century Ringer pointed out that calcium could be used in laboratory animals to counteract the toxic effects of potassium. Some investigators have recently suggested its intravenous use for patients with potassium poisoning. This method at best produces only a brief beneficial effect.

CALCIUM EXCESS

Calcium excess is always secondary to metabolic disease. A low calcium diet combined with the treatment of the underlying disease is the only satisfactory means of treating this excess. If patients with calcium excess require fluid therapy, calcium-free solutions should be used.

SUMMARY

1) Treatment of extracellular fluid volume excess varies with the cause of the imbalance and with the functional status of the body homeostatic mechanisms. Such measures as withholding of fluids, mercurial diuresis, phlebotomy, low sodium diet and improvement of nutrition may be indicated.

2) Treatment of potassium excess aims at removing excessive potassium from the extracellular fluid. Methods used include:

a) Elimination of potassium intake.
b) Administration of sodium chloride.
c) Administration of insulin and dextrose.
d) Treatment with testosterone.
e) Use of desoxycorticosterone acetate.
f) Extrarenal dialysis.
g) Administration of carbonic anhydrase inhibitors.
h) Use of ion exchange resins.
i) Intravenous administration of calcium.

3) Treatment of calcium excess consists of a low calcium diet plus management of the underlying disease.

Part IV Therapy: Changes in Position of Extracellular Fluid

29

When Fluid Shifts Occur

> *The physics of a man's circulation are the physics of the waterworks of the town in which he lives, but once out of gear, you cannot apply the same rules for the repair of the one as of the other.*
>
> Sir William Osler

Changes in the position of the extracellular fluid include:

1) Plasma-to-interstitial fluid shift.
2) Interstitial fluid-to-plasma shift.

Therapy of these shifts will be discussed in this chapter.

PLASMA-TO-INTERSTITIAL FLUID SHIFT

A plasma-to-interstitial fluid shift may be small and localized as seen in a sprained ankle, or it may be massive and diffuse as is seen in an extensive burn. It may be manifested by generalized edema or by a collection of fluid as seen in ascites. Fluid therapy is urgently needed when a large shift occurs rapidly. Such a shift is sometimes accompanied by shock because of the great decrease in plasma volume. Shock may be followed by complete vascular collapse and heart failure.

Therapy includes:

1) Limiting the shift if possible.
2) Maintaining plasma volume.
3) Treating vascular collapse and heart failure if they occur.
4) Preparing for the possibility of a secondary interstitial fluid-to-plasma shift.

Limiting the Shift

It is often not possible to restrict a massive shift except by treatment of the primary disease. A tight binder around the affected part may be helpful in localized shifts. Fortunately, even massive shifts are self-limited because of such factors as increased local tissue tension and depression of blood pressure.

Maintaining the Plasma Volume

The most satisfactory way to maintain plasma volume in the case of massive shifts is to supply plasma by the intravenous route. If plasma is not available, a plasma expander such as dextran can be used. If the shift involves only water and electrolytes and not plasma protein, fluids similar in concentration and composition to extracellular fluid should be used. No. 158 *gastrointestinal replacement solution* or lactated Ringer's solution is ideal if the kidneys are functioning. If renal depression is present, either isotonic solution of sodium chloride or saline lactate solution, described in Chapter 27, may be given. The amount of plasma, dextran or electrolyte solution to be used in an individual patient varies with the weight of the patient and the magnitude of the shift.

The physician has several sources of information to help guide him in the determination of the proper dose of plasma, dextran or electrolyte solution. First, the plasma volume is usually about 50 ml. per kilogram (2.2 pounds) of body weight. Rarely will more than half of this amount shift into the interstitial fluid. For this reason the maximal dose of the parenteral solution chosen will rarely exceed 25 ml. per kilogram (2.2 pounds) of body weight.

A second guide is the blood pressure, if heart failure or peripheral vascular collapse has not occurred. The systolic blood pressure should be maintained at as near normal as possible by administration of the fluids mentioned in the previous paragraphs.

In the absence of hemorrhage, repeated hemoglobin, hematocrit or plasma protein determinations help measure the degree of shift. In therapy the hemoglobin should be maintained between 10 and 13 Gm., and the hematocrit between 30 and 40% in infants and children under 3 years of age. The hemoglobin should be maintained between 11 and 15 Gm., and the hematocrit between 40 and 50% in older children and adults. The plasma protein level should be maintained between 6 and 8 Gm. per 100 ml. in patients of any age.

Urine flow is another helpful guide for regulation of therapy. Urinary output should be kept between 10 and 30 ml. per square meter of body surface per hour.

Vascular Collapse and Heart Failure

Vascular collapse and heart failure are almost always fatal complications. They are usually caused by prolonged anoxia secondary to shock. Their treatment includes administration of oxygen and stimulants such as epinephrine, nor-epinephrine, caffeine or coramine.

Secondary Interstitial Fluid-to-Plasma Shift

A secondary interstitial fluid-to-plasma shift can occur as a sequel to a plasma-to-interstitial fluid shift. If the physician is aware of this possibility, he will avoid *unnecessary* intravenous administration of fluids during a plasma-to-interstitial fluid shift. Such unnecessary expansion of plasma volume will set the stage for an overloading of the renocardiovascular system should a secondary interstitial fluid-to-plasma shift occur.

INTERSTITIAL FLUID-TO-PLASMA SHIFT

Recall that there are two classes of interstitial fluid-to-plasma shifts. The first class includes the rapid shift that occurs with remobilization of edema fluid following a plasma-to-interstitial fluid shift or that results from excessive intravenous administration of hypertonic solutions, plasma, serum albumin or dextran. The second class is a gradual compensatory shift that may result from internal or external loss of whole blood.

In regard to the first class of shift, spontaneous remobilization of edema fluid, such as may occur in burns, is not preventable. The shift that results from unwise intravenous administration of hypertonic solutions, plasma, serum albumin or dextran is, of course, avoidable.

If critical overloading of the vascular system does occur, the amount of blood returning to the heart should be reduced. This can be accomplished either by placing tourniquets around the extremities or by venous bleeding (phlebotomy). Tourniquets should be adjusted so as to obstruct the venous return, not the arterial circulation. If phlebotomy is performed, the initial bleeding should not exceed 10 ml. per kilogram (2.2 pounds) of body weight. The patient should be observed closely during the procedure. Red cells removed should be replaced or oral ferrous sulfate therapy should be instituted.

The compensatory shift that occurs because of loss of blood can be treated by transfusions of red blood cells, if severe, or by oral ferrous sulfate therapy or both.

SUMMARY

Plasma-to-Interstitial Fluid Shift

1) The goals of treatment of a plasma-to-interstitial fluid shift are two:

   a) Restrict the shift by relief of the condition causing the shift and by application of a binder for localized shifts.

   b) Maintain or restore plasma volume by parenteral administration of plasma, dextran or a plasma-like electrolyte solution.

2) The *maximal* dose of the parenteral solution chosen will rarely exceed 25 ml. per kilogram (2.2 pounds) of body weight.

3) Dose of solution and rate of administration should be such that:

   a) Systolic blood pressure will be maintained at a fairly normal level.

   b) Hemoglobin will be maintained between 10 and 13 Gm. per 100 ml. and the hematocrit between 30 and 40% in infants and young children.

   c) Hemoglobin will be maintained between 13 and 15 Gm. per 100 ml. and the hematocrit between 40 and 50% in older children and adults.

   d) Plasma protein level will be maintained between 6 and 8 Gm. per 100 ml. for patients of all ages.

   e) Urinary output will be kept between 10 and 30 ml. per square meter of body surface per hour.

4) Vascular collapse and heart failure of critical shifts are treated by oxygen and stimulants.

5) To avoid a secondary interstitial fluid-to-plasma shift, do not overtreat a plasma-to-interstitial fluid shift.

Interstitial Fluid-to-Plasma Shift

1) Treatment of an interstitial fluid-to-plasma shift resulting from remobilization of edema fluid consists of phlebotomy or application of tourniquets around the extremities.

2) Treatment of a shift caused by excessive intravenous administration of hypertonic solutions, plasma, serum albumin or dextran is usually unnecessary; in extreme cases, phlebotomy may be employed.

3) A shift resulting from internal or external loss of whole blood is treated by blood transfusion, if severe. After large external losses of blood, the patient should be given oral ferrous sulfate therapy.

Part V Therapy: Changes in Nutritional Status of Body

30

Nutritional Deficits

> *. . . Only food can feed;*
> *Not air, not dust, not water through a sieve.*
>
> ABBIE HUSTON EVANS

NUTRITIONAL STATUS changes most prone to occur in patients with prolonged illness include:

Protein deficit.
Caloric deficit.
Vitamin deficit.

This type of imbalance involves an imbalance both of fluid and solid constituents of the body. Therapy will be discussed in this chapter.

Nutritional deficits are frequently overlooked because they develop gradually. They present few dramatic clinical findings and precipitate no acute emergencies. Nevertheless, recovery of the patient frequently depends upon whether his nutritional status improves or deteriorates. The vicious cycle of disease, anorexia, malnutrition and further anorexia can be interrupted only by food by mouth, nasogastric tube or vein.

While the oral or nasogastric route is the best way to supply food, parenteral feeding should not be neglected. As Elman has so well emphasized, "Intravenous feeding competes only with starvation. In fact, in successfully combating starvation it restores or maintains the patient so much better that oral feeding can be started and increased much sooner."

PROTEIN DEFICIT

Protein starvation begins when protein intake is less than is required to meet the patient's minimal needs. Ingestion of food sources of protein may decrease gradually or cease abruptly, as in the case of the surgical patient who receives nothing by mouth. While protein deprivation for a day or two probably offers no hazard to the well nourished patient, careful laboratory determinations indicate a decrease in the plasma proteins within a few days after protein intake stops. Weech has shown that loss of 1 Gm. of circulating plasma protein reflects removal of about 30 Gm. of body protein. Each gram of body protein lost represents a loss of about 5 Gm. of protoplasm.

Protein is best given in a high protein diet rich in meat, eggs, cheese, fish and fowl. If the patient cannot ingest a normal diet, protein can be given in repeated small liquid high protein feedings or administered by polyvinyl nasogastric tube. When there is a contraindication to such feedings, such as intestinal obstruction, or when it is desired to supplement oral or tube feedings, protein can be administered intravenously as a solution of protein hydrolysate and carbohydrate. In referring to one such preparation, Albright said: "The effect of casein hydrolysate . . . on nitrogen metabolism is not materially altered by its route of administration, whether by vein or by mouth, and is essentially the same as that of an equivalent amount of beef protein by mouth."

In order to prevent the utilization of infused amino acids for energy purposes, it is advantageous to administer carbohydrate simultaneously, hence, solutions of protein hydrolysates usually contain dextrose, fructose or alcohol or combinations of these. It has been shown by repeated studies that it is *not* necessary to fully meet the body's caloric needs to prevent infused protein from being catabolized for energy.

Protein hydrolysates have the advantage of providing amino acids, the building blocks of protein; these are transported to the liver and other organs for prompt synthesis into needed body protein. *Neither blood nor plasma is suitable for replenishing the depleted protein stores of the body.*

In addition to providing amino acids, peptides and carbohydrate, protein hydrolysate solutions contain electrolytes in varying amounts, depending upon the preparation. One casein hydrolysate which has had its electrolyte composition adjusted so as to conform to the *balanced solutions* previously described has the following electrolyte composition in milliequivalents per liter:

cations	sodium	30
	potassium	15
	calcium	5
	magnesium	2
anions	chloride	22
	phosphate	30

Inspection reveals that there is a total of 52 mEq. of cations (or anions) per liter. Most protein hydrolysate solutions are so formulated as to contain 50 or 100 Gm. of amino acids and peptides per liter of solution. In addition, 50 or 100 Gm. of carbohydrate is usually incorporated in each liter of solution. Fifty grams of ethyl alcohol per liter, in addition to 50 Gm. of carbohydrate, is incorporated in one solution to provide additional calories and mild sedation.

Any protein hydrolysate solution with an electrolyte composition approximating that of the solution described can be given in place of an equal volume of a *balanced solution* when the patient's condition requires infusion of a source of protein nutrition.

CALORIC DEFICIT

Calories, which are derived chiefly from fat and carbohydrate, provide energy. They are required in greatly enhanced amounts during acute illness and convalescence. The average normal caloric intake is between 1500 and 2000 calories per square meter of body surface per day. If calories cannot be provided by mouth or tube, they should be given parenterally, as dextrose, fructose or alcohol. Intravenous infusion of fat is still in the experimental stage.

Any patient on an exclusively parenteral feeding regime should receive 60 to 75 Gm. of carbohydrate per square meter of body surface per day to minimize the ketosis and tissue catabolism associated with fasting.

Parenteral solutions, regardless of their type, usually contain 50 or 100 Gm. of dextrose or fructose per liter, corresponding to 5 or 10% solutions respectively. The choice of a 5 or 10% solution should rest upon the needs of the patient for calories and the total quantity of parenteral solutions which he will receive daily.

VITAMIN DEFICIT

Both clinical and experimental evidence indicate an increased need for vitamins during periods of stress such as acute illness, infectious disease, surgery, burns, injury and convalescence. The ideal source of vitamins is a well balanced diet. For patients unable to ingest such a diet, vitamins should be supplied from another source. Either liquid or capsule preparations can be given to many patients. Others may require parenteral administration of vitamins, either as concentrates or as constituents of parenteral solutions. Particularly important for the patient under stress are generous amounts of vitamins of the B complex and vitamin C.

A vitamin intake which has been suggested for the patient undergoing stress is as follows:

thiamine hydrochloride	10 mg.
riboflavin	10 mg.
niacinamide	100 mg.
calcium pantothenate	20 mg.
pyridoxine hydrochloride	2 mg.
folic acid	1.5 mg.
vitamin B_{12}	4 mcg.
ascorbic acid	300 mg.

This formulation will undoubtedly be modified from time to time.

SUMMARY

1) Recovery of the patient may depend upon his nutritional status.

2) Protein, the keystone nutrient, can be given as a high protein diet, as an oral or tube feeding, or intravenously as a solution of protein hydrolysate and carbohydrate. Several such solutions are actually *balanced solutions* from the standpoint of their electrolyte compositions.

3) Calories are required in greatly increased amounts in illness. The patient on an exclusively parenteral feeding regime should receive 60 to 75 Gm. of carbohydrate per square meter of body surface per day to meet his minimal caloric needs.

4) The need for vitamins is increased during periods of stress. While the ideal source of these nutritional essentials is a balanced diet, the sick patient should receive vitamins from other sources such as oral liquids or capsules or parenteral preparations.

Part VI Therapy: Routes of Administration

31

Which Road to Scotland

> *Oh! ye'll take the high road*
> *And I'll take the low road,*
> *And I'll be in Scotland afore ye; . . .*
> TRADITIONAL FOLK SONG

EACH OF THE several routes over which fluids may enter the body has its peculiar advantages, disadvantages, indications and contraindications. Whether the best road to the Scotland of optimal therapeutic effect is the high road, the low road, or perhaps a middle road depends upon considerations which will be presented in this chapter.

ENTERAL ROUTES

Enteral routes for the administration of fluids include all pathways for which some portion of the gastrointestinal tract serves as the portal.

Water is absorbed in the stomach, small intestine and large intestine. While electrolytes are absorbed primarily in the small intestine, some absorption occurs in the large intestine. The same can be said for carbohydrates. Both water and electrolytes are absorbed when administered rectally, particularly when the need is great and when the intestinal tract is not hyperactive.

The oral route is unquestionably the most effective and safe route for administration of water, electrolytes and nutrient substances, provided the patient's gastrointestinal tract is functioning properly and no contraindication exists. If the patient's gastrointestinal tract is functional but he is unwilling or unable to take fluids by mouth, they can be administered by the nasal route through polyvinyl nasogastric tubing.

Gastrostomies and enterostomies have been employed for administration of nutrient feedings. Cecostomies and colostomies are rarely used as a means of administering fluids.

The rectal route is not as satisfactory as the oral or the nasogastric route for fluid administration. Nevertheless, this route can be useful for the home management of patients who are vomiting or who have sustained mild fluid deficits because of fever or sweating.

Certain precautions are important in administering fluids by rectum. Before the infusion is begun, a cleansing enema should be given. The fluid can then be administered as a slow drip or by intermittent rectal instillation of small amounts; for example, 1, 2, or 3 ounces. If a slow drip is used, the reservoir containing the fluid should be suspended at a level of about six inches above the level of the anus. The rate of flow should be adjusted in accordance with the patient's ability to retain the fluid. Intermittent instillation of 1 or 2 ounces of fluid every one to two hours has been found to be useful in infants and small children.

Oral electrolyte mixtures are surprisingly well retained when administered by mouth, even in patients who are unable to retain formula, milk or other liquid feedings. Similarly, oral electrolyte mixtures have proved successful when administered rectally in infants and in children who were vomiting but in whom severe diarrhea was not present. *It is important in giving an oral electrolyte mixture to give it in the prescribed dilution.* Otherwise, excessive amounts of electrolytes may be absorbed, leading to the development of electrolyte concentration excess.

The limits of maximal tolerance have been exceeded in infants when oral electrolyte mixtures were added to the infant's formula or when excessively concentrated homemade mixtures were given.

PARENTERAL ROUTES

Three parenteral routes are utilized for the administration of water, electrolytes and nutrient substances. These include the subcutaneous route, the intravenous route and bone marrow infusions.

Subcutaneous Route

Fluids can be administered subcutaneously either as a continuous drip or by means of intermittent injections under pressure. The site most commonly employed for continuous drip is the thigh. Intermittent injections are usually given in the skin of the back between the scapulas. The amount of any fluid that can be administered subcutaneously varies with the size of the patient, the rate of absorption, the type of fluid used and the requirements of the patient. Hyaluronidase, an enzyme that temporarily breaks down the hyaluronic acid barriers of connective tissue, can be employed to accelerate the diffusion of fluids administered subcutaneously. Whether the rate of absorption is also accelerated has not been demonstrated.

The subcutaneous route has the advantage that it presents minimal technical difficulty and, hence, does not usually require the services of an intern or attending physician. Use of the subcutaneous route may, however, be dangerous, since fluids administered subcutaneously form a pool which must first be absorbed into the plasma before it can be utilized by the body. During the course of absorption, the body attempts to convert the subcutaneous pool of fluid into a fluid resembling extracellular fluid. If the subcutaneous pool differs greatly in electrolyte concentration or electrolyte composition from extracellular fluid, water or electrolytes will be drawn from the extracellular fluid into the subcutaneous pool. As a result of this shift, deficits in extracellular fluid volume, in electrolyte concentration or in electrolyte composition may develop or be intensified.

The following types of solutions are not satisfactory for subcutaneous administration:

1) Hypertonic solutions such as any 10% carbohydrate solution.
2) 5% carbohydrate solutions that also contain electrolytes; a 5% carbohydrate solution, in itself isotonic, is rendered hypertonic by additional electrolytes.
3) Carbohydrate-in-water solutions; these are hazardous because they contain no electrolytes.
4) Hypotonic solutions of any type.
5) Electrolyte solutions whose composition or concentration differs greatly from extracellular fluid.

The most satisfactory solutions for subcutaneous administration are those containing no carbohydrate and having an electrolyte concentration and composition similar to those of extracellular fluid. No. 158 *gastrointestinal replacement solution* and lactated Ringer's solution are satisfactory for subcutaneous administration provided they contain no carbohydrate. Such solutions cannot be employed to supply the daily water requirement because they provide no free water.

Intravenous Route

The intravenous route is by far the most satisfactory for the administration of parenteral fluids. Unfortunately, starting and maintaining an intravenous infusion is not always simple, particularly in infants and small children. Fluids administered intravenously at the proper rate and in the proper dose pass directly into the extracellular fluid; they are rapidly acted upon by the body homeostatic mechanisms, hence, do not produce abnormal changes in the volume, electrolyte concentration or electrolyte composition of the extracellular fluid. Any solution designed for parenteral administration can be given intravenously. Hypertonic solutions tend to be damaging to veins in direct proportion to their degree of hypertonicity. Such damage can be minimized by use of small needles, care in venipuncture and alternation of sites of venipuncture.

The antecubital vein of the arm is commonly used for intravenous infusions in larger children and adults. The scalp veins, veins of the wrist, and veins of the back of the hand are usually more accessible in infants. Superficial veins of the forearm and foot may be employed in either infants or adults. In emergencies and in situations in which a continuous infusion of several days duration is necessary, a so-called "cut-down" on the great saphenous vein of the ankle at the point where it passes anterior to the medial tibial malleolus may be necessary. This procedure represents a minor surgical operation which requires care and skill.

Bone Marrow Infusions

Bone marrow infusion of fluids may be hazardous and should be employed only when intravenous infusion has been unsuccessful.

Considerable amounts of water, electrolytes, plasma or blood can be administered by way of the bone marrow. The rate of absorption is slower than that with intravenous infusion.

The mid-tibia is the site of choice for bone marrow infusions for infants and small children. The manubrium of the sternum is the location of choice for larger children and adults.

A short, flat-bevel 18-gauge needle with a stylet is inserted using firm pressure until the bone is contacted. A rotary motion is then employed until the marrow is entered, as indicated by a sensation of decreased resistance. The stylet is then removed and a small amount of marrow withdrawn into a syringe to ascertain whether the end of the needle is in the marrow cavity. The infusion is then started.

The rate of bone marrow infusion should not exceed that used for intravenous infusion. The duration of the infusion should not exceed 24 hours. The same bone should not be used more than once every 48 hours because of the danger of osteomyelitis.

SUMMARY

Enteral Routes

1) Enteral routes of administration include the mouth, the nose and the rectum. Other enteral routes are surgical openings into the gastrointestinal tract.

2) The oral route is the enteral route of choice. The nasal route, through which nasogastric tubes are inserted, is second choice. The rectal route is third choice.

Parenteral Routes

1) Fluids can be administered parenterally by the subcutaneous route, the intravenous route and by bone marrow infusion.

2) The subcutaneous route requires minimal technical skill, but is suitable for only a limited number of solutions because of certain hazards. The safest solutions for subcutaneous administration are the No. 158 *gastrointestinal replacement solution* and lactated Ringer's solution.

3) The intravenous route may offer technical difficulties in in-

fants and small children, but is usually the parenteral route of choice. Fluids administered intravenously are promptly absorbed and exert a rapid therapeutic effect. Any solution designed for parenteral administration can be given intravenously.

4) Bone marrow infusions are of limited usefulness.

Part VII Down to Cases

32

Analysis of Clinical Cases

Science is a first-rate piece of furniture for a man's upper chamber, if he has common sense on the ground floor.

Oliver Wendell Holmes

What you have read up to this point of the handbook has provided you with all the information needed to understand the fluid balance diagnosis and therapy for the case histories in this chapter, and to work out your own diagnosis and therapy for the cases in the "workshop" of Chapter 33. Before proceeding with the case histories, the physician will do well to review Chapters 22 through 31. He will also be well advised to *memorize* the summaries of Chapters 22 and 26, particularly the rules for rate of administration.

To aid the physician in an orderly study of the body fluid status of a given patient, we have developed a *fluid imbalance analysis sheet* (Table 8). This is designed for use in conjunction with the *laboratory values useful in diagnosis* (Table 1, Chapter 14) and the *daily fluid balance sheet* (Table 2, Chapter 14).

To use the fluid imbalance analysis sheet, the physician records points from the history, physical examination and laboratory findings which suggest changes in the properties of extracellular fluid in the designated columns. In the space toward the bottom of the sheet he notes any changes in position of extracellular fluid and in the nutritional status of the patient.

A composite diagnosis of the fluid imbalances present can then be made and appropriate therapy planned and instituted. Such an analysis should be made as often as necessary, for example, every 24 hours, every 12 hours or even every 8 hours, depending upon the clinical progress of the patient.

In every case presented in this chapter, a complete history was taken and a physical examination was performed, following the method suggested in Chapter 14. However, only the *positive* findings of the history and physical examination are listed in the case histories. Results of all laboratory tests are presented.

As explained previously, variations in names applied by different manufacturers to their parenteral and oral solutions have made it necessary for us to employ either generally used or coined generic names in our discussions of fluid therapy. Representative commercial solutions corresponding to these generic names are presented in Table 28.

CASE 1. A 48-year-old female was admitted to the hospital for an elective hysterectomy. The history revealed that the menopause had occurred at the age of 45. The patient had complained of a feeling of increased pelvic pressure for several years.

Physical examination revealed
- enlarged uterus secondary to multiple myomata
- weight 62 Kg. (138 pounds)
- height 62 inches

Laboratory examination of the blood revealed
- hemoglobin 15 Gm.
- hematocrit 42%
- white blood count 8000 with a normal differential

Urinalysis revealed
- pH 6.0
- specific gravity 1.015
- otherwise negative

Clinical Progress

At operation the uterus was removed without difficulty. The postoperative course was smooth and parenteral fluids were not necessary after the first day.

The fluid balance diagnosis and therapy for the first postoperative day are summarized in Table 8.

Explanation of Management as Presented in Table 8

1) Body surface area: 1.63 square meters.

TABLE 8

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Multiple Uterine Myomata NAME OR NUMBER Case 1 SEX F AGE 48

Properties of Extracellular Fluid

	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Diagnosis	Normal	Normal	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Normal

Management

1) Body surface area: 1.63 square meters.
2) First infusion, begun following operation – No. 75 balanced solution containing 10% carbohydrate: 1200 ml. intravenously at rate of 6.5 ml. per minute in three hours and five minutes.
3) Second infusion, begun 12 hours after completion of operation – No. 75 balanced solution containing 10% carbohydrate: 1200 ml. intravenously at rate of 6.5 ml. per minute in three hours and five minutes.

(Body surface area was determined from nomogram using height and weight.)

2) First infusion, begun following operation—No. 75 *balanced solution* containing 10% carbohydrate: 1200 ml. intravenously at rate of 6.5 ml. per minute in three hours and five minutes.

(This infusion represented the first portion of the 2400 ml. of *balanced solution* calculated to supply water and electrolytes for maintenance at dosage level of 1500 ml. per square meter of body surface per day. Calculation: 1500 ml. × 1.63 = 2445 ml. or about 2400 ml.

(Rate of infusion of 6.5 ml. per minute based on 4 ml. per square meter of body surface per minute. Calculation: 4 ml. × 1.63 = 6.5 ml.)

3) Second infusion, begun 12 hours after completion of operation—No. 75 *balanced solution* containing 10% carbohydrate: 1200 ml. intravenously at rate of 6.5 ml. per minute in three hours and five minutes.

(This infusion represented the second portion of the 2400 ml. of *balanced solution.*)

COMMENT

This case illustrates the type of parenteral fluid therapy necessary to provide maintenance amounts of water and electrolytes to a patient who is not losing fluids except by way of the skin, lungs and kidneys, and who has not sustained any previous deficits. The *balanced solutions* when given at the dosage level of 1500 ml. per square meter of body surface per day provide the necessary water and electrolytes. The carbohydrate in the solutions provides for minimal caloric needs.

The *balanced solution* given was divided into two equal amounts which were started 12 hours apart. The rate of administration, 4 ml. per square meter of body surface per minute, was used to decrease the time of infusion and thus the discomfort of the patient.

CASE 2. An 8-year-old boy was admitted to the hospital because of vomiting and diarrhea of two days duration. He had not retained oral feedings for 36 hours and had not voided for 24 hours. He weighed about 60 pounds at the onset of the illness.

Physical examination revealed a lethargic child with

rectal temperature 101° F.

pulse 105

respirations 24 (deep and regular)
blood pressure 110/80
dry skin and mucous membranes
acetone odor to the breath
increase in intestinal peristaltic sounds
weight 25 Kg. (55 pounds)
height 50 inches

Laboratory examination of the blood revealed
hemoglobin 15 Gm.
plasma sodium 148 mEq./L.
plasma potassium 5.0 mEq./L.
plasma bicarbonate 10 mEq./L.
plasma chloride 108 mEq./L.

Examination of urine obtained by catheterization revealed
bladder urine volume 10 ml.
pH 5.0
specific gravity 1.032
trace of albumin
acetone present

Clinical Progress (First 24 Hours)

Nausea, vomiting and diarrhea persisted throughout the first day. Estimated fluid lost from vomiting and diarrhea was 500 ml. in the first 12 hours and 400 ml. during the second 12 hours.

The fluid balance diagnosis and management for the first 24 hours are summarized in Table 9.

Explanation of Management as Presented in Table 9

1) Body surface area: 0.97 square meters.

(Body surface area was determined from nomogram using height and weight.)

2) Single infusion—

a) No. 51 *pump priming solution* containing 10% carbohydrate: 234 ml. intravenously at rate of 7.8 ml. per minute in 30 minutes followed by

(*Pump priming solution* was used because voiding had not occurred for 24 hours. Dosage level of 360 ml. per square meter of body surface per 45 minutes was employed. Rate of infusion of 7.8 ml. per minute was based on 8 ml. per square meter of body surface per minute. Calculation: 8 ml. × 0.97 = 7.8 ml. After 234 ml. had been infused, patient voided.)

TABLE 9

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Gastroenteritis NAME OR NUMBER Case 2 SEX M AGE 8

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	1) Vomiting and diarrhea for 2 days 2) No fluid intake for 36 hours 3) Failure to void for 24 hours. Suggestive of deficit	1) Fever (increased loss of water from skin and lungs)Suggestive of excess	1) Diminished caloric intake 2) Failure to void for 24 hours. Suggestive of base bicarbonate deficit (metabolic acidosis) 1) Increased potassium losses 2) Inadequate intake. Suggestive of extracellular and cellular potassium deficit
Physical Examination	1) Dry skin and mucous membranes 2) Weight loss of 5 pounds (8.5% loss in body weight) Indicative of deficit	Negative for deficit or excess	1) Rapid deep respirations 2) Acetone odor of breath Suggestive of base bicarbonate deficit (metabolic acidosis)

Laboratory Findings	1) Hemoglobin 15 Gm. 2) Small urine volume on catheterization 3) Urine specific gravity 1.032 Indicative of deficit	1) Plasma sodium 148 mEq./L. 2) Hemoglobin 15 Gm. Indicative of excess	1) Plasma bicarbonate 10 mEq./L. 2) Acetone present in urine. Indicative of base bicarbonate deficit (metabolic acidosis)
Diagnosis	Severe fluid volume deficit	Electrolyte concentration excess	1) Base bicarbonate deficit (metabolic acidosis) 2) Probable potassium deficit

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Caloric deficit assumed because of history of no oral intake for 36 hours

Management

1) Body surface area: 0.97 square meters.
2) Single infusion--
    a) No. 51 pump priming solution containing 10% carbohydrate: 234 ml. intravenously at rate of 7.8 ml. per minute in 30 minutes followed by
    b) No. 75 balanced solution containing 10% carbohydrate: 2000 ml. intravenously at rate of 2.9 ml. per minute in 11 hours and 30 minutes followed by
    c) No. 75 balanced solution containing 10% carbohydrate: 1200 ml. intravenously at rate of 1.67 ml. per minute in second 12 hours.

b) No. 75 *balanced solution* containing 10% carbohydrate: 2000 ml. intravenously at rate of 2.9 ml. per minute in 11 hours and 30 minutes followed by

(This infusion represented the first portion of the 2700 ml. of *balanced solution* calculated to supply water and electrolytes for maintenance and to correct the severe fluid volume deficit at the dosage level of 3000 ml. per square meter of body surface per day. Calculation: 3000 ml. × 0.97 = 2910 ml. The 234 ml. of *pump priming solution* infused was subtracted from 2910 ml. to give 2676 ml. or about 2700 ml.

(Rate of infusion was 2.9 ml. per minute based on 3 ml. per square meter of body surface per minute. Calculation: 3 ml. × 0.97 = 2.9 ml.)

c) No. 75 *balanced solution* containing 10% carbohydrate: 1200 ml. intravenously at rate of 1.67 ml. per minute in second 12 hours.

(This infusion represented the remaining 700 ml. of *balanced solution* calculated to supply water and electrolytes for maintenance and to correct the severe fluid volume deficit. It also included 500 ml. of *balanced solution* necessary to replace fluid lost from diarrhea and vomiting during the first 12 hours. The slow infusion rate of 1.67 ml. per minute was used because of the probable necessity for continuing parenteral fluid therapy during the next 24 hours. Calculation: 1200 ml. divided by 720—the minutes in 12 hours— = 1.67 ml.)

Clinical Progress (Second 24 Hours)

Clinically, body fluid status appeared normal. Severe nausea precluded use of oral fluid. Diarrhea gradually subsided. In the first 12 hours 300 ml. of fluid was lost in diarrheal stools. In the second 12 hours 200 ml. of fluid was lost in diarrheal stools.

Fluid balance diagnosis and management for the second 24 hours are summarized in Table 10.

Explanation of Management as Presented in Table 10

1) Single infusion—

a) No. 75 *balanced solution* containing 10% carbohydrate: 1850 ml. intravenously at rate of 2.57 ml. per minute in first 12 hours followed by

(This infusion included the 1450 ml. of *balanced solution* calculated to supply water and electrolytes for maintenance at the dosage level of 1500 ml. per square meter of body surface per day. Calculation: 1500 ml. × 0.97 = 1450 ml.

(This infusion also included 400 ml. of *balanced solution* necessary for replacement of concurrent losses from diarrhea and vomiting dur-

TABLE 10

FLUID IMBALANCE ANALYSIS

Day 2

ADMISSION DIAGNOSIS Gastroenteritis NAME OR NUMBER Case 2 SEX M AGE 8

Properties of Extracellular Fluid

	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Diagnosis	Normal	Normal	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Management 1) Single infusion--

a) No. 75 balanced solution containing 10% carbohydrate: 1850 ml. intravenously at rate of 2.57 ml. per minute in first 12 hours followed by

b) No. 75 balanced solution containing 10% carbohydrate: 300 ml. intravenously at rate of 2.57 ml. per minute in next hour and 56 minutes.

ing the previous 12 hours. The slow fluid administration rate of 2.57 ml. per minute was employed because of the necessity of continuing the infusion into the next 12 hours. Calculation: 1850 ml. divided by 720—minutes in 12 hours— = 2.57 ml.)

b) No. 75 *balanced solution* containing 10% carbohydrate: 300 ml. intravenously at rate of 2.57 ml. per minute in next hour and 56 minutes.

(This infusion represented replacement of concurrent fluid loss from diarrhea of 300 ml. during the previous 12 hours. The infusion rate of 2.57 ml. per minute of the previous infusion was employed. After this infusion, intravenous fluids were discontinued.)

Clinical Progress (Third 24 Hours)

Clinically, body fluid status appeared normal. No nausea, vomiting or diarrhea occurred.

Fluid balance diagnosis and management for the third day are summarized in Table 11.

Explanation of Management as Presented in Table 11

1) Oral *balanced solution:* 700 ml. taken ad libitum in first 12 hours.

(This solution was given since the child was able to retain oral fluids.)

2) 800 ml. skim milk taken orally during the remainder of the day.

(Skim milk was given to provide protein and as a transition to solid foods.)

Comment

This case demonstrates the simultaneous occurrence of several types of specific fluid imbalances in a severe gastroenteritis. Changes in properties of extracellular fluid included:

1) volume deficit because of fluid lost through vomiting, diarrhea and insensible perspiration at the time when fluid intake was inadequate,
2) electrolyte concentration excess due to a disproportionate loss of water in comparison to electrolytes,
3) base bicarbonate deficit (metabolic acidosis) due to ketosis and decreased urinary excretion of nonbicarbonate anions, and
4) probable extracellular and cellular potassium deficit secondary to losses which exceeded intake.

TABLE 11

FLUID IMBALANCE ANALYSIS

Day 3

ADMISSION DIAGNOSIS Gastroenteritis NAME OR NUMBER Case 2 SEX M AGE 8

Properties of Extracellular Fluid

	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Diagnosis	Normal	Normal	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Management 1) Oral balanced solution: 700 ml. taken ad libitum in first 12 hours.
2) 800 ml. skim milk taken orally during the remainder of the day.

The fluid volume deficit was corrected by the water and electrolytes in the parenteral *balanced solution*. The concentration excess was corrected by the free water available in the *balanced solution*. Base bicarbonate deficit (metabolic acidosis) was corrected because the parenteral *balanced solution* supplied:

1) lactate ion to support the base bicarbonate side of the carbonic acid: base bicarbonate ratio,
2) water to help eliminate accumulated nonbicarbonate anions, and
3) carbohydrate as a fat-free source of energy to avoid ketone body formation.

Potassium deficit was corrected by potassium contained in the *balanced solution*. Parenteral fluid therapy was not necessary after the second 24 hours.

On the third day oral *balanced solution* was offered ad libitum for 12 hours. Skim milk was then started. A full diet was gradually introduced in the next two days and the child was discharged from the hospital on the fifth day.

CASE 3. A 33-year-old man was brought to the hospital because of collapse at work. History revealed that he had worked for six hours on a road construction project while the outside temperature was above 90° F. and the relative humidity about 65%. His fellow workers stated he had perspired profusely and ingested about three or four quarts of water during this period.

Physical examination revealed a pale, lethargic man with
- oral temperature 99° F.
- pulse 95
- blood pressure 80/40
- weight 85 Kg. (187 pounds)
- height 72 inches

Laboratory examination of the blood revealed
- hemoglobin 14 Gm.
- plasma sodium 125 mEq./L.
- plasma chloride 90 mEq./L.
- plasma bicarbonate 25 mEq./L.

Urinalysis revealed
- pH 6.0
- specific gravity 1.002
- otherwise negative

Clinical Progress

The patient responded well to treatment and was able to leave the hospital the following day.

The fluid balance diagnosis and management are summarized in Table 12.

Explanation of Management as Presented in Table 12

1) Body surface area: 2.06 square meters.

(Body surface area was determined from nomogram using height and weight.)

2) First infusion—3% sodium chloride solution: 212 ml. intravenously at rate of 2 ml. per minute in one hour and 46 minutes.

(No fluid volume deficit was present, therefore, 3% sodium chloride solution was employed. The approximate amount of solution required to correct the deficit was 423 ml. Calculation: Plasma sodium deficit per liter of extracellular fluid = 142 mEq./L. minus 125 mEq./L. = 17 mEq./L. Approximate extracellular fluid weight = 85 Kg. × 0.15 =12.75 Kg. Since one kilogram of extracelluar fluid equals approximately one liter, patient's extracellular fluid volume is 12.75 liters. Sodium deficit in extracellular fluid = 12.75 × 17 mEq. = 217 mEq. of sodium.

(The total deficit of sodium is 217 mEq. There are 513 mEq. of sodium in 1000 ml. of 3% sodium chloride solution. The volume of 3% sodium chloride solution required to repair the deficit will, therefore, be the fraction 217/513 of 1000 ml. The volume of solution is found thus: $\frac{217 \times 1000 \text{ ml.}}{513} = 423$ **ml.**

(The first infusion consisted of approximately one-half of the calculated dose of 3% sodium chloride solution. Rate of infusion of 2 ml. per minute based on 1 ml. per square meter of body surface per minute. Calculation: 1 ml. × 2.06 = about 2 ml. per minute.)

3) Redetermination of plasma sodium level after previous infusion: 132 mEq./L.

(Since the plasma sodium level on redetermination was still depressed below normal, the second half of the calculated dose was given as the second infusion.)

4) Second infusion—3% sodium chloride solution: 212 ml. intravenously at rate of 2 ml. per minute in one hour and 46 minutes.

(This was the remaining portion of the calculated dose of 3% sodium chloride solution.)

TABLE 12

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Moderate Heat Exhaustion NAME OR NUMBER Case 3 SEX M AGE 33

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	1) Profuse sweating combined with drinking of water 2) Collapse. Suggestive of deficit	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	1) Pallor 2) Pulse 95 3) Blood pressure 80/40 4) Temperature 99°F. (0) Suggestive of deficit	Negative for deficit or excess

Laboratory Findings	Negative for deficit or excess	1) Plasma sodium 125 mEq./L. Indicative of deficit	Negative for deficit or excess
Diagnosis	Normal	Electrolyte concentration deficit	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Normal

Management

1) Body surface area: 2.06 square meters.
2) First infusion--3% sodium chloride solution: 212 ml. intravenously at rate of 2 ml. per minute in one hour and 46 minutes.
3) Redetermination of plasma sodium level after previous infusion: 132 mEq./L.
4) Second infusion--3% sodium chloride solution: 212 ml. intravenously at rate of 2 ml. per minute in one hour and 46 minutes.

Comment

This case illustrates an electrolyte concentration deficit of the extracellular fluid resulting from replacement of water, sodium and chloride lost in the sweat with water alone. The patient did not have a fluid volume deficit, therefore, a hypertonic (3%) solution of sodium chloride was used to correct the electrolyte concentration deficit.

CASE 4. A 2-year-old girl was admitted to the hospital one hour after a generalized convulsive seizure. History revealed chronic "constipation" for which the mother was in the habit of giving two or three enemas a week. Because of a particularly intractable period of constipation, the mother gave 10 tap water enemas in the previous two days. The last enema was given three hours before admission. Increasing lethargy was noted six hours before admission. The child urinated frequently during the two-day period. She normally weighed about 29 pounds (13 Kg.)

Physical examination revealed a semistuporous child with
- rectal temperature 99° F.
- pulse 115
- respirations 14
- blood pressure 120/90
- suggestion of bilateral papilledema
- fingerprinting of skin
- weight 14 Kg. (31 pounds)
- height 34 inches

Laboratory examination of the blood revealed
- hemoglobin 9 Gm.
- plasma sodium 120 mEq./L.
- plasma chloride 80 mEq./L.
- plasma bicarbonate 25 mEq./L.

Urinalysis revealed
- pH 6.0
- specific gravity 1.002
- otherwise negative

The cerebrospinal fluid was normal

Clinical Progress

There was a dramatic response to therapy. Large volumes of urine were excreted during the next few hours. The child was discharged from the hospital 24 hours after admission, and at the time of discharge her weight was again 29 pounds (13 Kg.).

The fluid balance diagnosis and management are summarized in Table 13.

Explanation of Management as Presented in Table 13

1) Body surface area: 0.54 square meters.

(**Body surface area was determined from nomogram using height and weight.**)

2) First infusion—3% sodium chloride solution: 42 ml. intravenously at rate of 0.54 ml. per minute in one hour and 18 minutes.

(**No fluid volume deficit was present, therefore, 3% sodium chloride solution was employed. The approximate amount of solution required to correct the deficit was 84 ml. Calculation: Plasma sodium deficit per liter of extracellular fluid = 142 mEq./L. minus 120 mEq./L. = 22 mEq./L. Approximate extracellular fluid weight = 13 Kg. × 0.15 = 1.95 Kg. Since one kilogram of extracellular fluid equals approximately one liter, patient's extracellular fluid volume is 1.95 liters. Sodium deficit in extracellular fluid = 1.95 × 22 mEq. = 43 mEq. of sodium.**

(**The total deficit of sodium is 43 mEq. There are 513 mEq. of sodium in 1000 ml. of 3% sodium chloride solution. The volume of 3% sodium chloride solution required to repair the deficit will, therefore, be the fraction 43/513 of 1000 ml. The volume of solution is found thus:**

$$\frac{43 \times 1000 \text{ ml.}}{513} = 84 \text{ ml.}$$

(**The first infusion consisted of approximately one-half of the calculated dose of 3% sodium chloride solution. Rate of infusion of 0.54 ml. per minute based on 1 ml. per square meter of body surface per minute. Calculation: 1 ml. × 0.54 = 0.54 ml. per minute.**)

3) Redetermination of plasma sodium level after previous infusion: 130 mEq./L.

(**Since the plasma sodium level on redetermination was still depressed below normal, the second half of the calculated dose was given as the second infusion.**)

4) Second infusion—3% sodium chloride solution: 42 ml. intravenously at rate of 0.54 ml. per minute in one hour and 18 minutes.

(**This was the remaining portion of the calculated dose of 3% sodium chloride solution.**)

Comment

This case demonstrates an electrolyte concentration deficit of the extracellular fluid secondary to the washing out of electrolytes

TABLE 13

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Convulsion - Cause Unknown NAME OR NUMBER Case 4 SEX F AGE 2

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	1) Ten tap water enemas in 48 hours. Suggestive of excess	1) Ten tap water enemas in 48 hours. Suggestive of deficit	Negative for deficit or excess
Physical Examination	1) Weight gain of 2 pounds. Indicative of excess	1) Temperature 99°F.(R) 2) Pulse 115 3) Blood pressure 120/90 4) Poor skin turgor 5) Fingerprinting of skin. Suggestive of deficit	Negative for deficit or excess

Laboratory Findings	1) Hemoglobin 9 Gm. Suggestive of excess	1) Plasma sodium 120 mEq./L. 2) Plasma chloride 80 mEq./L. 3) Urine specific gravity 1.002. Indicative of deficit	Negative for deficit or excess
Diagnosis	Fluid volume excess	Electrolyte concentration deficit	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Normal

Management
1) Body surface area: 0.54 square meters.
2) First infusion--3% sodium chloride solution: 42 ml. intravenously at rate of 0.54 ml. per minute in one hour and 18 minutes.
3) Redetermination of plasma sodium level after previous infusion: 130 mEq./L.
4) Second infusion--3% sodium chloride solution: 42 ml. intravenously at rate of 0.54 ml. per minute in one hour and 18 minutes.

by repeated tap water enemas. The fluid volume excess resulted because water was absorbed more rapidly from the enemas than it could be excreted by the kidneys. The fluid volume excess contributed to the electrolyte concentration deficit.

The electrolyte concentration deficit of the extracellular fluid was corrected by a 3% sodium chloride solution. Diuresis during and following the infusion of the hypertonic sodium chloride solution indicated correction of the fluid volume excess.

CASE 5. A 6-month-old boy was admitted to the hospital with a history of a "cold" for one week and high fever for three days. He had not eaten solid food since the onset of the fever, and had taken only a few sips of water during the 24 hours before admission. He last voided 12 hours before admission. He weighed 17 pounds at the onset of the illness.

Physical examination revealed a child in opisthotonus with
rectal temperature 104° F.
pulse 120
respirations 40
dry skin and mucous membranes
surprisingly good tissue turgor
rhinopharyngitis
bilateral acute otitis media
coarse rhonchi in both lungs
weight 7 Kg. (15.5 pounds)
height 27 inches

Laboratory examination of the blood revealed
hemoglobin 14 Gm.
white blood count 18,000 with 69% polymorphonuclear cells
plasma sodium 152 mEq./L.
plasma potassium 5.5 mEq./L.
plasma chloride 115 mEq./L.
plasma bicarbonate 10 mEq./L.

Urinalysis of a specimen voided 30 minutes after onset of fluid therapy revealed
pH 5.5
specific gravity 1.014
trace of albumin
acetone present

The cerebrospinal fluid was normal.

Clinical Progress

The combined effect of fluid, antipyretic and antibiotic therapy produced a remarkable change in 24 hours. The patient was able to take adequate fluid orally after the first day and was discharged on the fifth day.

Fluid balance diagnosis and management for the first 24 hours are summarized in Table 14.

Explanation of Management as Presented in Table 14

1) Body surface area: 0.36 square meters.

(Body surface area was determined from nomogram using height and weight.)

2) Single infusion—

a) No. 51 *pump priming solution* containing 10% carbohydrate: 87 ml. intravenously at rate of 2.9 ml. per minute in 30 minutes followed by

(*Pump priming solution* was used because voiding had not occurred for 12 hours. Dosage level of 360 ml. per square meter of body surface per 45 minutes was employed. Rate of infusion of 2.9 ml. per minute was based on 8 ml. per square meter of body surface per minute. Calculation: 8 ml. × 0.36 = 2.9 ml. After 87 ml. had been administered, the patient voided.)

b) No. 75 *balanced solution* containing 10% carbohydrate: 777 ml. intravenously at rate of 0.55 ml. in the next 23 hours and 30 minutes.

(This infusion represented the quantity of *balanced solution* calculated to supply water and electrolytes for maintenance and to correct the moderate fluid volume deficit at the dosage level of 2400 ml. per square meter of body surface per day. Calculation: 2400 ml. × 0.36 = 864 ml. minus 87 ml. = 777 ml.

(Slow infusion rate of 0.55 ml. per minute was used because of the possible necessity for continuing parenteral fluid therapy after the first 24 hours. Calculation: 777 ml. divided by 1410—the minutes in 23 hours and 30 minutes— = 0.55 ml.)

Comment

This case illustrates an electrolyte concentration excess of the extracellular fluid as it often manifests itself in young infants. Frequently, an initial diagnosis of meningeal disease is made. The electrolyte concentration excess results from the combination of

TABLE 14

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Upper Respiratory Infection – Possibly Meningitis NAME OR NUMBER Case 5 SEX M AGE 6 mo.

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	1) Inadequate fluid intake for at least 24 hours 2) Failure to void for 12 hours. Suggestive of deficit	1) Poor water intake for 24 hours 2) Fever for three days (increased water losses from skin and lungs). Suggestive of excess	1) Inadequate caloric intake for at least 24 hours 2) Failure to void for 12 hours. Suggestive of base bicarbonate deficit (metabolic acidosis)
Physical Examination	1) Weight loss 1.5 pounds (9% body weight loss) Indicative of deficit	1) Opisthotonus (evidence of nervous system stimulation) 2) Elevated temperature and rapid respirations (increased water losses from skin and lungs) Suggestive of excess	1) Rapid respirations Suggestive of base bicarbonate deficit (metabolic acidosis)

Laboratory Findings	1) Hemoglobin 14 Gm. (normal for this age 11 Gm.) Indicative of deficit	1) Plasma sodium 152 mEq./L. 2) Plasma chloride 115 mEq./L. Indicative of excess	1) Plasma bicarbonate 10 mEq./L. 2) Acetone present in urine. Indicative of base bicarbonate deficit (metabolic acidosis)
Diagnosis	Moderate fluid volume deficit	Electrolyte concentration excess	Base bicarbonate deficit (metabolic acidosis)

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Caloric deficit suggested by inadequate caloric intake for at least 24 hours.

Management

1) Body surface area: 0.36 square meters.
2) Single infusion--
    a) No. 51 pump priming solution containing 10% carbohydrate: 87 ml. intravenously at rate of 2.9 ml. per minute in 30 minutes followed by
    b) No. 75 balanced solution containing 10% carbohydrate: 777 ml. intravenously at rate of 0.55 ml. per minute in the next 23 hours and 30 minutes.

decreased water intake and increased water loss from skin and lungs. In this patient electrolyte concentration excess was associated with fluid volume deficit and base bicarbonate deficit (metabolic acidosis). The latter resulted from ketosis and decreased excretion of nonbicarbonate anions by the kidney.

The *pump priming solution* and the *balanced solution* supplied free water for correction of the electrolyte concentration excess and for elimination by the kidney of accumulated nonbicarbonate anions. The lactate in the *balanced solution* supported the base bicarbonate side of the carbonic acid: base bicarbonate ratio. The *balanced solution* also supplied water and electrolytes to meet maintenance needs and to correct the fluid volume deficit. Carbohydrate in the solutions provided a fat-free source of energy to help eliminate ketosis.

CASE 6. A 35-year-old man was admitted to the hospital because of recurrent pyloric obstruction secondary to a duodenal ulcer. The patient had vomited "everything he ate" for two days. On admission, gastric suction was started and during the first 36 hours 1500 ml. of gastric secretion was removed. Two liters of an isotonic solution of sodium chloride and two liters of a carbohydrate-in-water solution were infused during this period. The patient became increasingly lethargic and complained of a tingling sensation in the skin of his face, hands and feet. A consultant was called in because the patient was not responding to treatment. The results of physical and laboratory examinations done at this time, 36 hours after admission, follow.

Physical examination revealed a thin individual lying quietly in bed with physical findings of

oral temperature 98.6° F.
pulse 75
blood pressure 105/65
respirations 6 (shallow and irregular)
gastric suction tube in place
weight 61 Kg. (135 pounds)
height 68 inches

Laboratory examination of the blood revealed

hemoglobin 14 Gm.
hematocrit 45%
white blood count 9500 with a normal differential
plasma sodium 142 mEq./L.

plasma potassium 3.0 mEq./L.
plasma chloride 85 mEq./L.
plasma bicarbonate 40 mEq./L.

Urinalysis revealed
pH 7.0
specific gravity 1.012
otherwise negative

Clinical Progress (First 24 Hours)

Lethargy, tingling sensations in the skin, and hypoventilation disappeared with a change in the type of parenteral fluids administered. Gastric suction was continued throughout this 24-hour period. In the first 12 hours 700 ml. of gastric secretion was removed and 650 ml. in second 12 hours.

Fluid balance diagnosis and management for the first 24 hours are summarized in Table 15.

Explanation of Management as Presented in Table 15

1) Body surface area: 1.71 square meters.

(Body surface area was determined from nomogram using height and weight.)

2) First infusion—No. 75 *balanced solution* containing 10% carbohydrate: 1500 ml. intravenously at rate of 6.8 ml. per minute in three hours and 45 minutes.

(This infusion represented the first portion of the 2500 ml. of *balanced solution* calculated to supply water and electrolytes for maintenance at dosage level of 1500 ml. per square meter of body surface per day. Calculation: 1500 ml. × 1.71 = 2565 ml. or about 2500 ml. Rate of infusion of 6.8 ml. per minute based on 4 ml. per square meter of body surface per minute. Calculation: 4 ml. × 1.71 = 6.8 ml.)

3) Second infusion, begun 12 hours after start of first infusion—

a) Protein hydrolysate 5% in 5% carbohydrate solution: 1000 ml. intravenously at rate of 6.8 ml. per minute in two hours and 30 minutes followed by

(This solution is a *balanced solution* as well as a source of amino acids. The quantity of solution given supplied the remainder of the water and electrolytes for maintenance and helped repair the protein deficit.)

b) No. 150 *gastrointestinal replacement solution* containing 10% carbohydrate: 700 ml. intravenously at rate of 5.1 ml. per minute in two hours and 15 minutes.

TABLE 15

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Obstructed Duodenal Ulcer NAME OR NUMBER Case 6 SEX M AGE 35

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	1) Thirty-six hours of gastric suction 2) No potassium intake 3) Increasing lethargy 4) Tingling sensations Suggestive of base bicarbonate excess (metabolic alkalosis) and potassium deficit
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	1) Infrequent, irregular and shallow respirations. Suggestive of base bicarbonate excess (metabolic alkalosis)

Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	1) Plasma potassium 3.0 mEq./L. 2) Plasma chloride 85 mEq./L. 3) Plasma bicarbonate 40 mEq./L. Indicative of base bicarbonate excess (metabolic alkalosis) and potassium deficit
Diagnosis	Normal	Normal	Base bicarbonate excess (metabolic alkalosis) and potassium deficit

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Protein and caloric deficit suggested by absent oral intake of food

Management

1) Body surface area: 1.71 square meters.
2) First infusion--No. 75 balanced solution containing 10% carbohydrate: 1500 ml. intravenously at rate of 6.8 ml. per minute in three hours and 45 minutes.
3) Second infusion, begun 12 hours after start of first infusion--
    a) Protein hydrolysate 5% in 5% carbohydrate solution: 1000 ml. intravenously at rate of 6.8 ml. per minute in two hours and 30 minutes followed by
    b) No. 150 gastrointestinal replacement solution containing 10% carbohydrate: 700 ml. intravenously at rate of 5.1 ml. per minute in two hours and 15 minutes.

(This solution was administered to replace 700 ml. of gastric secretion removed by gastric suction in first 12 hours. Rate of infusion of 5.1 ml. per minute based on 3 ml. per square meter of body surface per minute. Calculation: 3 ml. × 1.71 = 5.1 ml.)

Clinical Progress (Second 24 Hours)

Clinically, the patient's body fluid status was normal. Redetermination of blood chemical values revealed

plasma potassium 4.0 mEq./L.
plasma chloride 95 mEq./L.
plasma bicarbonate 30 mEq./L.

Gastric suction was continued. In the first 12 hours 750 ml. of gastric secretion was removed and 600 ml. in the second 12 hours. A gastric resection was performed the next day. This report terminates just prior to operation.

Fluid balance diagnosis and management for the second 24 hours are summarized in Table 16.

Explanation of Management as Presented in Table 16

1) First infusion—

a) No. 75 *balanced solution* containing 10% carbohydrate: 1500 ml. intravenously at rate of 6.8 ml. per minute in three hours and 45 minutes followed by

(This infusion represented the first portion of the 2500 ml. of *balanced solution* calculated to supply water and electrolytes for maintenance at dosage level of 1500 ml. per square meter of body surface per day.)

b) No. 150 *gastrointestinal replacement solution* containing 10% carbohydrate: 650 ml. intravenously at rate of 5.1 ml. per minute in about two hours.

(This solution replaced gastric secretions removed in previous 12-hour period.)

2) Second infusion, begun 12 hours after start of first infusion—

a) Protein hydrolysate 5% in 5% carbohydrate solution: 1000 ml. intravenously at rate of 6.8 ml. per minute in two hours and 30 minutes followed by

(This solution is a *balanced solution* as well as a source of amino acids. The quantity of solution given supplied the remainder of the water and electrolytes for maintenance and helped repair the protein deficit.)

b) No. 150 *gastrointestinal replacement solution* containing 10%

carbohydrate: 750 ml. intravenously at rate of 5.1 ml. per minute in about two hours and 30 minutes.

(This solution was administered to replace 750 ml. of fluid removed by gastric suction in the first 12 hours.)

Comments

This case illustrates the persistence of a base bicarbonate excess (metabolic alkalosis) despite the use of isotonic solution of sodium chloride when potassium deficit is present. While both potassium and chloride were lost in gastric secretions, during the first 36 hours after hospital admission potassium was not replaced.

During the two day period prior to surgery, a *balanced solution,* which contained potassium, was administered. In addition, No. 150 *gastrointestinal replacement solution,* containing therapeutic amounts of potassium and chloride, was used to replace fluid obtained by gastric suction. On both days, a protein hydrolysate solution with an electrolyte content similar to *balanced solutions* provided part of the water and electrolytes for maintenance. The use of this latter solution compensated in part for the protein deficit. Carbohydrate in all of the solutions provided calories.

CASE 7. An 18-year-old female, known to be diabetic, was admitted to the hospital because of uncontrolled diabetes mellitus. History revealed development of sore throat and fever two days before admission. Coincidentally, she began to "spill" sugar in her urine. The day before admission she ingested little except water but continued on her usual insulin dose. During the eight hours before admission she complained of abdominal pain, vomited several times and became increasingly lethargic. She had urinated at frequent intervals.

Physical examination revealed a drowsy young woman with
rectal temperature 103° F.
pulse 110
respirations 30 (Kussmaul type)
blood pressure 110/75
acetone odor to the breath
dry skin and mucous membranes
acute exudative pharyngitis
weight 52.5 Kg. (115 pounds; usual weight about 120 pounds)
height 64 inches

TABLE 16

FLUID IMBALANCE ANALYSIS

Day 2

ADMISSION DIAGNOSIS Obstructed Duodenal Ulcer NAME OR NUMBER Case 6 SEX M AGE 35

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	1) Plasma potassium 4.0 mEq./L. 2) Plasma chloride 95 mEq./L. 3) Plasma bicarbonate 30 mEq./L. Indicative of mild base bicarbonate excess (metabolic alkalosis)

Diagnosis	Normal	Normal	Mild base bicarbonate excess (metabolic alkalosis)
Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.	Protein deficit suggested by absent oral intake of food.		
Management	1) First infusion-- a) No. 75 balanced solution containing 10% carbohydrate: 1500 ml. intravenously at rate of 6.8 ml. per minute in three hours and 45 minutes followed by b) No. 150 gastrointestinal replacement solution containing 10% carbohydrate: 650 ml. intravenously at rate of 5.1 ml. per minute in about two hours. 2) Second infusion, begun 12 hours after start of first infusion-- a) Protein hydrolysate 5% in 5% carbohydrate solution: 1000 ml. intravenously at rate of 6.8 ml. per minute in two hours and 30 minutes followed by b) No. 150 gastrointestinal replacement solution containing 10% carbohydrate: 750 ml. intravenously at rate of 5.1 ml. per minute in about two hours and 30 minutes.		

Laboratory examination of the blood revealed
hemoglobin 17 Gm.
hematocrit 48%
white blood count 20,000 with 85% polymorphonuclear cells
blood sugar 300 mg./100 ml.
plasma sodium 138 mEq./L.
plasma potassium 5.0 mEq./L.
plasma chloride 98 mEq./L.
plasma bicarbonate 8 mEq./L.

Urinalysis revealed
pH 5.5
specific gravity 1.035
4 plus sugar
acetone present
acetoacetic acid present

Clinical Progress

The patient responded well to appropriate doses of insulin, antibiotics and fluids. Oral fluids were started 12 hours after the beginning of therapy at which time intravenous fluids were discontinued. Concurrent fluid losses during the first 12 hours from increased insensible perspiration, sensible perspiration and polyuria were estimated to be 1000 ml. No appreciable concurrent fluid losses occurred thereafter. The patient required no further specific fluid therapy and was discharged from the hospital one week after admission.

Fluid balance diagnosis and management for the first 24 hours are summarized in Table 17.

Explanation of Management as Presented in Table 17

1) Body surface area: 1.56 square meters.

(Body surface area was determined from nomogram using height and weight.)

2) Single infusion—No. 75 *balanced solution* containing 10% fructose: 3400 ml. intravenously at rate of 4.7 ml. per minute in first 12 hours.

(This solution represented the major portion of the 3750 ml. of *balanced solution* calculated to provide water and electrolytes for maintenance and to correct the moderate fluid volume deficit at a dosage level of 2400 ml. per square meter of body surface per day. Calculation: 2400 ml. × 1.56 = 3744 ml. or about 3750 ml.

(After 3400 ml. of this solution was given, the patient was able to take oral fluids so intravenous fluid therapy was discontinued. Rate of

infusion of 4.7 ml. per minute was based on 3 ml. per square meter of body surface per minute. Calculation: 3 ml. × 1.56 = 4.7 ml.)

3) Oral *balanced solution* offered in amount of 150 ml. per hour. Total of 1350 ml. taken and retained in next nine hours.

(The oral *balanced solution* retained included 350 ml. necessary to complete the water and electrolytes required for maintenance and to correct the moderate fluid volume deficit. It also included the 1000 ml. required to replace concurrent fluid losses from insensible perspiration, sensible perspiration and polyuria during the previous 12 hours.)

Comment

This case illustrates the use of *balanced solutions* in a patient with diabetic acidosis. Such patients usually have a fluid volume deficit combined with base bicarbonate deficit (metabolic acidosis). The latter is caused for the most part by ketosis.

During the recovery phase, a pronounced extracellular fluid potassium deficit often occurs when potassium-free solutions are used. This deficit is the result of:

1) Expansion of the extracellular fluid with dilution of its potassium.
2) Transfer of potassium from the extracellular fluid to the cells as cellular glycogen is formed, and
3) Losses of potassium in the urine before institution of fluid therapy.

A low plasma phosphorus level occurs at the same time for the same reasons.

The *balanced solutions,* both parenteral and oral, provided water and electrolytes necessary for maintenance and for correction of the fluid volume deficit. They also provided therapeutic amounts of potassium and phosphorus.

The base bicarbonate side of the carbonic acid: base bicarbonate ratio was supported by lactate in the parenteral *balanced solution* and by lactate and citrate in the oral *balanced solution.* Insulin used in treating the diabetes helped eliminate ketosis by promoting proper utilization of blood sugar and thus eliminating the necessity for using body fat as an energy source.

Some physicians prefer to administer carbohydrate-free *bal-*

TABLE 17

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Diabetic Acidosis NAME OR NUMBER Case 7 SEX F AGE 18

Properties of Extracellular Fluid

	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	1) Poor fluid intake for 24 hours 2) Voiding, despite poor fluid intake Suggestive of deficit	Negative for deficit or excess	1) Past history of diabetes mellitus 2) Inadequate food intake for 24 hours Suggestive of base bicarbonate deficit (metabolic acidosis)
Physical Examination	1) Dry skin and mucous membranes 2) Fever (increased fluid loss from skin and lungs) 3) Possible weight loss of 5 pounds (4% loss of body weight). Indicative of deficit	Negative for deficit or excess	1) Kussmaul type respirations 2) Acetone odor to breath Suggestive of base bicarbonate deficit (metabolic acidosis)

Laboratory Findings	1) Hemoglobin 17 Gm. 2) Hematocrit 48%. Indicative of deficit	Negative for deficit or excess	1) Plasma bicarbonate 8 mEq./L. 2) Acetone present in the urine 3) Acetoacetic acid present in urine. Indicative of base bicarbonate deficit (metabolic acidosis)
Diagnosis	Moderate fluid volume deficit	Normal	Base bicarbonate deficit (metabolic acidosis)

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Caloric deficit suggested by poor food intake and uncontrolled diabetes.

Management
1) Body surface area: 1.56 square meters.
2) Single infusion--No. 75 balanced solution containing 10% fructose: 3400 ml. intravenously at rate of 4.7 ml. per minute in first 12 hours.
3) Oral balanced solution offered in amount of 150 ml. per hour. Total of 1350 ml. taken and retained in next nine hours.

anced solutions to patients with diabetic acidosis during the first few hours of treatment.

CASE 8. A 6-week-old boy was admitted to the hospital because of persistent vomiting for three weeks. History revealed projectile vomiting for one week and little fluid retention for two days. Despite the vomiting the child seemed hungry. The vomitus had never been bile stained. The patient had no stools for three days and had not voided for 12 hours. He weighed 8 pounds at birth, 10 pounds two weeks before admission, and 8 pounds 8 ounces the day before admission.

Physical examination revealed an emaciated male infant with
rectal temperature 99° F.
pulse 120
respirations 6 (shallow and irregular)
dry skin and mucous membranes
poor tissue turgor
large upper abdominal peristaltic waves
palpable pyloric tumor
weight 3.6 Kg. (8 pounds)
height 24 inches

Laboratory examination of the blood revealed
hemoglobin 15 Gm.
plasma bicarbonate 40 mEq./L.
plasma chloride 80 mEq./L.

Urinalysis performed on a specimen obtained 25 minutes after start of fluid therapy revealed
pH 6.9
specific gravity 1.018
acetone present
otherwise negative

Clinical Progress (First 24 Hours)

A "cut-down" was performed on the great saphenous vein of the ankle because of the probability of prolonged parenteral fluid therapy. The clinical status of body fluids improved greatly during the first 24 hours of parenteral fluid therapy.

Fluid balance diagnosis and management for the first 24 hours are summarized in Table 18.

Explanation of Management as Presented in Table 18

1) Body surface area: 0.27 square meters.

(Body surface area was determined from nomogram using height and weight.)

2) Single infusion—

a) No. 51 *pump priming solution* containing 10% carbohydrate: 52 ml. intravenously at rate of 2.1 ml. per minute in approximately 25 minutes followed by

(*Pump priming solution* was used because voiding had not occurred for 12 hours. Dosage level of 360 ml. per square meter of body surface per 45 minutes was employed. Rate of infusion of 2.1 ml. per minute was based on 8 ml. per square meter of body surface per minute. Calculation: 8 ml. × 0.27 = 2.1 ml. After 52 ml. had been infused, patient voided.)

b) No. 90 *balanced solution* containing 10% carbohydrate: 758 ml. intravenously at rate of 0.54 ml. per minute during remainder of 24-hour period.

(This solution represented the *balanced solution* required to provide water and electrolytes for maintenance and to correct the severe fluid volume deficit at the dosage level of 3000 ml. per square meter of body surface per day. Calculation: 3000 ml. × 0.27 = 810 ml. From this was subtracted 52 ml. of *pump priming solution*, giving 758 ml.

(The slow infusion rate of 0.54 ml. per minute was used because of the necessity for continuing parenteral fluid therapy over the entire 24-hour period. Calculation: 758 ml. divided by 1415—the minutes in 23 hours and 35 minutes— = 0.54 ml.)

Clinical Progress (12-Hour and 32-Minute Interval Prior to and During Operation)

The clinical status of the child's body fluids was considered normal 24 hours after admission. An additional 12-hour period elapsed before a Fredet-Ramstedt operation was performed for relief of the pyloric stenosis. The operative procedure was completed in 32 minutes.

Fluid balance diagnosis and management for this 12-hour preoperative period and the 32-minute operative period are summarized in Table 19.

Explanation of Management as Presented in Table 19

1) Single infusion—

a) No. 90 balanced solution containing 10% carbohydrate: 202 ml. intravenously at rate of 0.28 ml. per minute in 12 hours followed by

(This chart covers only 12 hours plus the 32-minute operative period, since it was desired to start a fresh 24-hour period of record immediately after operation. The *balanced solution* administered was calculated to provide water and electrolytes for maintenance at a

TABLE 18

FLUID IMBALANCE ANALYSIS

Day 1 (First 24 Hours)

ADMISSION DIAGNOSIS Hypertrophic Pyloric Stenosis NAME OR NUMBER Case 8 SEX M AGE 6 weeks

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	1) Failure to void for 12 hours 2) Prolonged vomiting 3) Recent weight loss Suggestive of deficit	Negative for deficit or excess	1) Prolonged vomiting Suggestive of base bicarbonate excess (metabolic alkalosis) and potassium deficit
Physical Examination	1) Dry skin and mucous membranes 2) Poor tissue turgor 3) Weight same as birth weight. Suggestive of deficit	Negative for deficit or excess	1) Slow shallow respirations. Suggestive of base bicarbonate excess (metabolic alkalosis)

Laboratory Findings	1) Hemoglobin 15 Gm. Indicative of deficit	Negative for deficit or excess	1) Plasma bicarbonate 40 mEq./L. 2) Plasma chloride 80 mEq./L. 3) Urine pH 6.9 Indicative of base bicarbonate excess (metabolic alkalosis)
Diagnosis	Severe fluid volume deficit	Normal	Base bicarbonate excess (metabolic alkalosis) and potassium deficit

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Protein and caloric deficit suggested by weight loss

Management

1) Body surface area: 0.27 square meters.
2) Single infusion--
   a) No. 51 pump priming solution containing 10% carbohydrate: 52 ml. intravenously at rate of 2.1 ml. per minute in approximately 25 minutes followed by
   b) No. 90 balanced solution containing 10% carbohydrate: 758 ml. intravenously at rate of 0.54 ml. per minute during remainder of 24-hour period.

TABLE 19

FLUID IMBALANCE ANALYSIS

Day 2 (12-Hour and 32-Minute Interval Prior to and During Operation)

ADMISSION DIAGNOSIS Hypertrophic Pyloric Stenosis NAME OR NUMBER Case 8 SEX M AGE 6 weeks

Properties of Extracellular Fluid

	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Diagnosis	Normal	Normal	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Protein and caloric deficit suggested by weight loss.

Management 1) Single infusion--
a) No. 90 balanced solution containing 10% carbohydrate: 202 ml. intravenously at rate of 0.28 ml. per minute in 12 hours followed by
b) No. 51 pump priming solution containing 10% carbohydrate: 9 ml. intravenously at rate of 0.28 ml. per minute during 32-minute operative period.

dosage level of 1500 ml. per square meter of body surface per day. Calculation: 1500 ml. × 0.27 = 405 ml. for 24 hours. Only one-half of 405 ml. or about 202 ml. was required for the 12-hour period. The slow rate of infusion of 0.28 ml. per minute was used because it was desired to administer fluids over the entire 12-hour period. Calculation: 202 ml. divided by 720—the minutes in 12 hours— = 0.28 ml.)

b) No. 51 *pump priming solution* containing 10% carbohydrate: 9 ml. intravenously at rate of 0.28 ml. per minute during 32-minute operative period.

(*Pump priming solution* was used during the operative period for two reasons. First, since it is a potassium-free solution the possibility of potassium excess was avoided should a sudden fall in blood pressure or renal insufficiency occur during the operation. Second, continuation of the intravenous infusion during the operative period assured availability of a vein should an emergency arise.

(The slow rate of infusion was the same as that used for administration of the *balanced solution.*)

Clinical Progress (First Postoperative 24 Hours)

The patient tolerated the surgical procedure well. Oral fluids were started six hours after the operation was completed. However, major fluid needs were met parenterally because of the small volume of oral intake and occurrence of vomiting.

Fluid balance diagnosis and management for the first postoperative day are summarized in Table 20.

Explanation of Management as Presented in Table 20

1) Single infusion—No. 90 *balanced solution* containing 10% carbohydrate: 405 ml. intravenously at rate of 0.28 ml. per minute in 24 hours.

(This solution represented the *balanced solution* calculated to provide water and electrolytes for maintenance at the dosage level of 1500 ml. per square meter of body surface per day. The slow infusion rate of 0.28 ml. per minute was used because it was desired to administer parenteral fluids over the entire 24-hour period.)

2) Oral *balanced solution,* begun six hours after operation: 8 ml. by mouth. Alternated every three hours with dilute milk formula. Quantity offered increased 4 ml. every feeding.

(The feedings actually offered every three hours, beginning six hours after operation, were: 8 ml. oral *balanced solution;* 12 ml. dilute milk formula; 16 ml. oral *balanced solution;* 20 ml. dilute milk formula; 24 ml. oral *balanced solution;* 28 ml. dilute milk formula.)

TABLE 20

FLUID IMBALANCE ANALYSIS

Day 3 (First Postoperative 24 Hours)

ADMISSION DIAGNOSIS Hypertrophic Pyloric Stenosis NAME OR NUMBER Case 8 SEX M AGE 6 weeks

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Diagnosis	Normal	Normal	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Protein and caloric deficit suggested by weight loss.

Management
1) Single infusion--No. 90 balanced solution containing 10% carbohydrate: 405 ml. intravenously at rate of 0.28 ml. per minute in 24 hours.
2) Oral balanced solution, begun six hours after operation: 8 ml. by mouth. Alternated every three hours with dilute milk formula. Quantity offered increased 4 ml. every feeding.

Clinical Progress (Second Postoperative 24 Hours)

Beginning with the second postoperative day, a milk-with-added-carbohydrate formula was given. Intravenous fluids were discontinued 12 hours after start of this day because patient was retaining all oral fluids given.

Fluid balance diagnosis and management for the second postoperative 24 hours are summarized in Table 21.

Explanation of Management as Presented in Table 21

1) Single infusion—No. 90 *balanced solution* containing 10% carbohydrate: 202 ml. intravenously at rate of 0.28 ml. per minute in 12 hours.

(This infusion represented a portion of the *balanced solution* calculated to provide water and electrolytes for maintenance at a dosage level of 1500 ml. per square meter of body surface per day. After 202 ml. had been infused during the first 12 hours, it became apparent that the patient was retaining the milk-with-added-carbohydrate formula which had been offered every three hours throughout the 12-hour period. Intravenous fluids were, therefore, discontinued.)

2) Formula of milk-with-added-carbohydrate: 240 ml. by mouth at rate of 30 ml. every three hours during entire 24-hour period.

(The child retained all the formula offered, a total of 240 ml. This amount served to provide the remainder of the water and electrolytes required for maintenance.)

Comment

This case illustrates the type of fluid therapy necessary to correct fluid imbalances associated with a hypertrophic pyloric stenosis. In most patients, a base bicarbonate excess (metabolic alkalosis) develops because of loss of chloride through vomiting. This excess is associated with potassium deficit and will not respond to therapy unless potassium is supplied. Inability to retain oral fluid leads to moderate or severe fluid volume deficit. A severe protein and caloric deficit is often present by the time a diagnosis is made. *Surgery should not be performed until the fluid imbalances have been at least partially corrected.*

A *pump priming solution* was given initially because of history of not voiding for 12 hours before admission to hospital. A *balanced solution* provided necessary chloride and potassium for the preoperative correction of base bicarbonate excess (metabolic alkalosis) and potassium deficit. It also provided water and electrolytes

TABLE 21

FLUID IMBALANCE ANALYSIS

Day 4 (Second Postoperative 24 Hours)

ADMISSION DIAGNOSIS Hypertrophic Pyloric Stenosis NAME OR NUMBER Case 8 SEX M AGE 6 weeks

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Diagnosis	Normal	Normal	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Protein and caloric deficit suggested by weight loss.

Management

1) Single infusion--No. 90 balanced solution containing 10% carbohydrate: 202 ml. intravenously at rate of 0.28 ml. per minute in 12 hours.
2) Formula of milk-with-added-carbohydrate: 240 ml. by mouth at rate of 30 ml. every three hours during entire 24-hour period.

for correction of the fluid volume deficit. Carbohydrate contained in all of the solutions contributed calories. Protein and caloric deficits can be completely corrected by oral feeding only, once the pyloric stenosis has been relieved.

Postoperative parenteral fluid therapy is usually necessary for 36 to 48 hours. On the first postoperative day, it is ordinarily necessary to supply all fluid needs parenterally because of restriction of oral intake and occurrence of vomiting. On the second postoperative day, about half of the fluid requirements are usually supplied by the parenteral route. The remainder can be met by the oral route.

The patient made an uneventful recovery and was discharged seven days postoperatively weighing 10 pounds.

CASE 9. An 8-month-old female was admitted to the hospital for the first of a series of plastic procedures designed to correct a congenital hare lip and cleft palate. Following operation the child was given 700 ml. of isotonic solution of sodium chloride during a 24-hour period. Because of generalized edema, a consultant was asked to see the child at the beginning of the second postoperative day. The patient's preoperative weight was 6.3 Kg. (13.8 pounds) and her preoperative hemoglobin was 11 Gm.

Physical examination revealed a child in no acute distress with
rectal temperature 99.8° F.
pulse 110
respirations 22
hare lip and cleft palate
generalized mild pitting edema
weight 6.8 Kg. (14.9 pounds)
height 28 inches

Laboratory examination of the blood revealed
hemoglobin 9.0 Gm.

No urine specimen was obtained

Clinical Progress

The child urinated frequently during the day. No evidence of heart failure or pulmonary edema developed. The edema had nearly disappeared within 24 hours after completion of the infusion of isotonic solution of sodium chloride.

Fluid balance diagnosis and management are summarized in Table 22.

TABLE 22

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Congenital Hare Lip and Cleft Palate NAME OR NUMBER Case 9 SEX F AGE 8 mo.

Properties of Extracellular Fluid

	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	1) Intravenous infusion of 700 ml. of isotonic solution of sodium chloride in 24 hours. Suggestive of excess	Negative for deficit or excess	1) Restricted potassium intake 2) Excessive sodium intake. Suggestive of potassium deficit
Physical Examination	1) Generalized pitting edema 2) Weight gain of 1.1 pounds. Indicative of excess	Negative for deficit or excess	Negative for deficit or excess

Laboratory Findings	1) Hemoglobin of 9.0 Gm. in comparison to preoperative hemoglobin of 11 Gm. Indicative of excess	Negative for deficit or excess	Negative for deficit or excess
Diagnosis	Fluid volume excess	Normal	Probable potassium deficit

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Normal

Management
1) Body surface area: 0.34 square meters.
2) Oral *balanced solution* was offered ad libitum. A total of 800 ml. was taken and retained.

Explanation of Management as Presented in Table 22

1) Body surface area: 0.34 square meters.

(Body surface area was determined from nomogram using height and preoperative weight.)

2) Oral *balanced solution* was offered ad libitum. A total of 800 ml. was taken and retained.

(The solution was given to the patient in accordance with her desires, since there was no serious impairment of body homeostasis.)

Comment

This case demonstrates development of an extracellular fluid volume excess as a result of excessive parenteral administration of isotonic solution of sodium chloride. The fluid volume excess was manifested by edema, weight gain and a fall in hemoglobin. Injudicious administration of isotonic solution of sodium chloride to postsurgical patients frequently results in retention of fluids.

No specific therapeutic measures were undertaken to correct the fluid volume excess because

1) there was no evidence of heart failure or pulmonary edema, and
2) The patient's homeostatic mechanisms were not seriously impaired and could be expected to correct the fluid volume excess.

The oral *balanced solution* provided free water for replacement of insensible losses by way of the skin and lungs. In addition, it supplied free water with which the patient's kidneys could excrete some of the excess sodium chloride. Potassium in the oral *balanced solution* aided in the replacement of potassium lost because of the combined effect of restricted potassium intake and excessive sodium intake. Carbohydrate in the oral *balanced solution* provided necessary calories.

The patient's edema completely subsided in 36 hours. The remainder of the postoperative course was uneventful.

CASE 10. A 29-year-old rancher was admitted to the hospital because of severe abdominal pain. History revealed intermittent attacks of epigastric distress over a five-year period. Distress was relieved by food. One week before admission the patient experienced the onset of another attack. Three hours before admission his epigastric pain increased in severity and became generalized over the abdomen.

Physical examination revealed a young man in pain with
oral temperature 99° F.
pulse 115
respirations 25
blood pressure 90/50
moderate abdominal distention
tenderness of the entire abdominal wall to palpation
board-like rigidity of the abdominal wall
absent bowel sounds
weight 82 Kg. (180 pounds)
height 72 inches

Laboratory examination of the blood revealed
hemoglobin 16 Gm.
hematocrit 45%
white blood count 18,000 with 70% polymorphonuclear cells

A catheterized urine specimen revealed
bladder urine volume 75 ml.
pH 5.8
specific gravity 1.030
trace of albumin
otherwise negative

Roentgenogram of the abdomen in the erect position revealed free air beneath the diaphragm.

Clinical Progress (First 24 Hours)

The patient had a perforated duodenal ulcer. Because of shock, norepinephrine was administered preoperatively until the patient's blood pressure was stable. The perforated ulcer was surgically repaired one hour after admission to the hospital. About 500 ml. of turbid fluid was removed from the abdomen at the time of operation. Because adynamic ileus secondary to chemical peritonitis developed postoperatively, continuous gastric suction was employed. The fluid obtained by suction was bile stained and had a pH of 6.9. It was presumed to be a mixture of gastric and small intestinal secretions. During the first 12 hours 800 ml. of suction fluid was obtained and 600 ml. during the second 12 hours.

Fluid balance diagnosis and management for the first 24 hours are summarized in Table 23.

Explanation of Management as Presented in Table 23

1) Body surface area: 2.02 square meters.

(Body surface area was determined from nomogram using height and weight.)

TABLE 23

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Perforated Peptic Ulcer NAME OR NUMBER Case 10 SEX M AGE 29

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Diagnosis	Normal	Normal	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Evidence of shock, chemical peritonitis and fluid in the peritoneal cavity indicative of internal pooling of extracellular fluid.

Management

1) Body surface area: 2.02 square meters.
2) First infusion--
   - a) No. 51 pump priming solution containing 10% carbohydrate with 4 ml. per liter of nor-epinephrine added: 60 ml. intravenously at rate of 2 ml. per minute in 30 minutes followed by
   - b) No. 51 pump priming solution containing 10% carbohydrate: 124 ml. intravenously at rate of 4 ml. per minute during 31 minutes required for operative procedure followed by
   - c) No. 75 balanced solution containing 10% carbohydrate, begun after completion of operation: 1300 ml. intravenously at rate of 8 ml. per minute in two hours and 42 minutes followed by
   - d) No. 158 gastrointestinal replacement solution containing 10% carbohydrate: 500 ml. intravenously at rate of 6 ml. per minute in one hour and 24 minutes.
3) Second infusion, begun 12 hours after completion of operation--
   - a) No. 75 balanced solution containing 10% carbohydrate: 1500 ml. intravenously at rate of 8 ml. per minute in three hours and seven minutes followed by
   - b) No. 158 gastrointestinal replacement solution containing 10% carbohydrate: 400 ml. intravenously at rate of 6 ml. per minute in one hour and six minutes followed by
   - c) No. 150 gastrointestinal replacement solution containing 10% carbohydrate: 400 ml. intravenously at rate of 6 ml. per minute in one hour and six minutes.

2) First infusion—

a) No. 51 *pump priming solution* containing 10% carbohydrate with 4 ml. per liter of nor-epinephrine added: 60 ml. intravenously at rate of 2 ml. per minute in 30 minutes followed by

(*Pump priming solution* with nor-epinephrine added was given to combat shock, the solution being employed merely as a convenient carrier for the nor-epinephrine. The slow infusion rate of 2 ml. per minute corresponded to the recommended rate of administration for solutions containing nor-epinephrine in the concentration used.)

b) No. 51 *pump priming solution* containing 10% carbohydrate: 124 ml. intravenously at rate of 4 ml. per minute during 31 minutes required for operative procedure followed by

(*Pump priming solution* was used during the operative period for two reasons. First, since it is a potassium-free solution the possibility of potassium excess was avoided should a sudden fall in blood pressure or renal insufficiency occur during operation. Second, continuation of the intravenous infusion during the operative period assured availability of a vein should an emergency arise.

(The 124 ml. of *pump priming solution* was given during the surgical procedure at the rate of 4 ml. per minute. Calculation: 2 ml. × 2.02 = 4 ml. This slow rate of infusion is the same as that used when *balanced solutions* are given in a single infusion for maintenance.)

c) No. 75 *balanced solution* containing 10% carbohydrate, begun after completion of operation: 1300 ml. intravenously at rate of 8 ml. per minute in two hours and 42 minutes followed by

(This solution was the first portion of the 2800 ml. of *balanced solution* calculated to provide water and electrolytes for maintenance at the dosage level of 1500 ml. per square meter of body surface per day. Calculation: 1500 ml. × 2.02 = 3030 ml. Subtracting the 60 ml. of *pump priming solution* containing nor-epinephrine given before operation and the 124 ml. of *pump priming solution* given during operation equals 2846 ml., or about 2800 ml.

(Rate of infusion of 8 ml. per minute based on 4 ml. per square meter of body surface per minute. Calculation: 4 ml. × 2.02 = 8 ml.)

d) No. 158 *gastrointestinal replacement solution* containing 10% carbohydrate: 500 ml. intravenously at rate of 6 ml. per minute in one hour and 24 minutes.

(This solution replaced 500 ml. of approximately isotonic fluid removed from the peritoneal cavity at the time of operation. Infusion rate of 6 ml. per minute was based on 3 ml. of body surface per minute. Calculation: 3 ml. × 2.02 = 6 ml.)

3) Second infusion, begun 12 hours after completion of operation—

a) No. 75 *balanced solution* containing 10% carbohydrate: 1500 ml. intravenously at rate of 8 ml. per minute in three hours and seven minutes followed by

(This solution represented the second half of the daily maintenance requirement for water and electrolytes.)

b) No. 158 *gastrointestinal replacement solution* containing 10% carbohydrate: 400 ml. intravenously at rate of 6 ml. per minute in one hour and six minutes followed by

(This solution replaced half of the fluid obtained by suction during the first 12 hours after operation. Because the suction fluid had a pH of 6.9, it was assumed to contain equal amounts of gastric and intestinal secretions. Hence, it was replaced half by No. 158 *gastrointestinal replacement solution* containing 10% carbohydrate, and half by No. 150 *gastrointestinal replacement solution* containing 10% carbohydrate.)

c) No. 150 *gastrointestinal replacement solution* containing 10% carbohydrate: 400 ml. intravenously at rate of 6 ml. per minute in one hour and six minutes.

(This infusion replaced the other half of the fluid obtained by suction during the first 12 hours after the operation.)

Clinical Progress (First 24 Hours)

Clinically, the patient's body fluid status was normal. During the first 12 hours 400 ml. of fluid similar to that obtained on the first day was removed by gastric suction. Bowel sounds returned late on the second day and suction was then discontinued.

Fluid balance diagnosis and management for the second 24 hours are summarized in Table 24.

Explanation of Management as Presented in Table 24

1) First infusion—

a) No. 75 *balanced solution* containing 10% carbohydrate: 1500 ml. intravenously at rate of 8 ml. per minute in three hours and seven minutes followed by

(This solution provided half of the 3000 ml. of *balanced solution* calculated to provide water and electrolytes for daily maintenance.)

b) No. 158 *gastrointestinal replacement solution* containing 10% carbohydrate: 300 ml. intravenously at rate of 6 ml. per minute in 50 minutes followed by

(As explained above, the suction fluid was estimated to contain equal amounts of gastric and intestinal secretions. This solution replaced one-half of the suction fluid obtained during the previous 12-hour period.)

TABLE 24

FLUID IMBALANCE ANALYSIS

Day 2

ADMISSION DIAGNOSIS Perforated Peptic Ulcer NAME OR NUMBER Case 10 SEX M AGE 29

Properties of Extracellular Fluid

	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Diagnosis	Normal	Normal	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Normal

Management

1) First infusion--
    - a) No. 75 balanced solution containing 10% carbohydrate: 1500 ml. intravenously at rate of 8 ml. per minute in three hours and seven minutes followed by
    - b) No. 158 gastrointestinal replacement solution containing 10% carbohydrate: 300 ml. intravenously at rate of 6 ml. per minute in 50 minutes followed by
    - c) No. 150 gastrointestinal replacement solution containing 10% carbohydrate: 300 ml. intravenously at rate of 6 ml. per minute in 50 minutes.
2) Second infusion, begun 12 hours after start of first infusion--
    - a) No. 75 balanced solution containing 10% carbohydrate: 1500 ml. intravenously at rate of 8 ml. per minute in three hours and seven minutes followed by
    - b) No. 158 gastrointestinal replacement solution containing 10% carbohydrate: 200 ml. intravenously at rate of 6 ml. per minute in 33 minutes followed by
    - c) No. 150 gastrointestinal replacement solution containing 10% carbohydrate: 200 ml. intravenously at rate of 6 ml. per minute in 33 minutes.

c) No. 150 *gastrointestinal replacement solution* containing 10% carbohydrate: 300 ml. intravenously at rate of 6 ml. per minute in 50 minutes.

(This solution replaced the second half of the suction fluid obtained during the previous 12-hour period.)

2) Second infusion, begun 12 hours after start of first infusion—

a) No. 75 *balanced solution* containing 10% carbohydrate: 1500 ml. intravenously at rate of 8 ml. per minute in three hours and seven minutes followed by

(This solution represented the second half of the 3000 ml. of *balanced solution* calculated to meet the daily maintenance requirement.)

b) No. 158 *gastrointestinal replacement solution* containing 10% carbohydrate: 200 ml. intravenously at rate of 6 ml. per minute in 33 minutes followed by

(This solution replaced one-half of the suction fluid obtained during the previous 12-hour period.)

c) No. 150 *gastrointestinal replacement solution* containing 10% carbohydrate: 200 ml. intravenously at rate of 6 ml. per minute in 33 minutes.

(This solution replaced the second half of the suction fluid obtained during the previous 12-hour period.)

Comment

This case illustrates the fluid therapy necessary for a postoperative patient who requires gastric or duodenal suction for several days.

In this particular patient, shock, as evidenced by tachycardia and fall in blood pressure, was present at the time of admission. The shock was for the most part secondary to pain and fear. Shift of plasma into the peritoneal cavity secondary to chemical peritonitis may have contributed to the shock. The shock was treated by control of pain with a narcotic, and the intravenous administration of nor-epinephrine in a *pump priming solution*. This solution was prepared by the addition of 4 ml. of the commercial preparation of nor-epinephrine to a liter of *pump priming solution*. Thirty minutes after start of the infusion, the patient's blood pressure was stabilized at 120/80.

Balanced solutions provided water and electrolytes for maintenance. Fluid lost by gastric suction was replaced by equivalent amounts of *gastrointestinal replacement solutions*. One-half was replaced by No. 150 *gastrointestinal replacement solution* and one-half by No. 158 *gastrointestinal replacement solution* because the suction fluid was a mixture of gastric and intestinal secretions.

CASE 11. A 29-year-old female was admitted to the hospital because of abdominal pain. History revealed nausea and epigastric pain of 36 hours duration. The pain gradually migrated to the right lower abdomen. Vomiting had occurred several times. Fluid intake consisted of a few ounces of water and a bowl of broth. Patient complained of thirst. No stools had been passed since the onset of the pain. Patient had last voided more than eight hours before admission.

Physical examination revealed
- oral temperature 100.6° F.
- pulse 98
- respirations 22
- blood pressure 116/75
- dry skin and mucous membranes
- fair tissue turgor
- tenderness and rigidity of the abdomen, maximal in the right lower quadrant
- rebound abdominal tenderness
- absent bowel sounds
- weight 54.7 Kg. (120 pounds)
- height 64 inches

Laboratory examination of the blood revealed
- hemoglobin 16 Gm.
- hematocrit 47%
- white blood count 16,000 with 95% polymorphonuclear cells

Analysis of a catheterized urine specimen revealed
- bladder urine volume 75 ml.
- pH 5.5
- specific gravity 1.032
- trace of albumin
- acetone present
- occasional red and white blood cells found on microscopic examination

Clinical Progress (First 24 Hours)

A diagnosis of acute appendicitis was made. At operation, the appendix was gangrenous and ruptured. Abscess formation had begun. The appendix was removed and the abscess drained. Antibiotics were started. A suction tube was inserted in the duodenum postoperatively because of an adynamic ileus. During the first 12 hours 450 ml. suction fluid was obtained and 375 ml. during the second 12 hours.

Fluid balance diagnosis and management for the first 24 hours are summarized in Table 25.

Explanation of Management as Presented in Table 25

1) Body surface area: 1.56 square meters.

(Body surface area was determined from nomogram using height and weight.)

2) First infusion—

a) No. 51 *pump priming solution* containing 10% carbohydrate: 250 ml. intravenously at rate of 12.5 ml. per minute in 20 minutes followed by

***(Pump priming solution* was used because voiding had not occurred for at least eight hours. Dosage level of 360 ml. per square meter of body surface per 45 minutes was used. Rate of infusion of 12.5 ml. per minute was based on 8 ml. per square meter of body surface per minute. Calculation: 8 ml. × 1.56 = 12.5 ml. This was given prior to operation. After 250 ml. had been infused, patient voided.)**

b) No. 51 *pump priming solution* containing 10% carbohydrate: 100 ml. intravenously at rate of 3.1 ml. per minute during 32 minutes required for operative procedure followed by

***(Pump priming solution* was used during the operative period for two reasons. First, since it is a potassium-free solution the possibility of potassium excess was avoided should a sudden fall in blood pressure or renal insufficiency occur during operation. Second, continuation of the intravenous infusion during the operative period assured availability of a vein should an emergency arise.**

(The 100 ml. of *pump priming solution* was given during the surgical procedure at the rate of 3.1 ml. per minute. Calculation: 2 ml. × 1.56 = 3.1 ml. This slow rate of infusion is the same as that used when *balanced solutions* are given in a single infusion for maintenance.)

c) No. 90 *balanced solution* containing 10% carbohydrate: 2000 ml. intravenously at rate of 4.7 ml. per minute in seven hours and six minutes.

(This solution was the first portion of the 3400 ml. of *balanced solution* calculated to provide water and electrolytes for maintenance and for correction of the moderate fluid volume deficit at the dosage level of 2400 ml. per square meter of body surface per day. Calculation: 2400 ml. × 1.56 = 3744 ml. Subtracting the 250 ml. of *pump priming solution* given prior to operation and the 100 ml. of *pump priming solution* given during operation from 3744 equals 3394 or about 3400 ml.

(Rate of infusion of 4.7 ml. per minute based on 3 ml. per square meter of body surface per minute. Calculation: 3 ml. × 1.56 = 4.7 ml.

(Intravenous fluids were then discontinued for six hours for the comfort of the patient.)

3) Second infusion, begun approximately 14 hours after completion of operation—

a) No. 90 *balanced solution* containing 10% carbohydrate: 1400 ml. intravenously at rate of 4.7 ml. per minute in five hours followed by

(This solution completed the daily dose of *balanced solution* above calculated.)

b) No. 158 *gastrointestinal replacement solution* containing 10% carbohydrate: 450 ml. intravenously at rate of 4.7 ml. per minute in one hour and 36 minutes.

(This solution replaced the 450 ml. of intestinal secretions obtained by suction during the previous 12 hours.)

Clinical Progress (Second 24 Hours)

Clinically, the patient's body fluid status was normal. Only parenteral fluids were given on this day. Bowel sounds returned late on the second day. Intestinal suction was then discontinued. During the first 12 hours 350 ml. of suction fluid was obtained.

Fluid balance diagnosis and management for the second 24 hours are summarized in Table 26.

Explanation of Management as Presented in Table 26

1) First infusion—

a) No. 90 *balanced solution* containing 10% carbohydrate: 1200 ml. intravenously at rate of 6.2 ml. per minute in three hours and 14 minutes followed by

(This solution was the first portion of the 2400 ml. of *balanced solution* calculated to provide water and electrolytes for maintenance at a dosage level of 1500 ml. per square meter of body surface per day. Calculation: 1500 ml. × 1.56 = 2340 ml. or about 2400 ml.

(Rate of infusion of 6.2 ml. per minute was based on 4 ml. per square meter of body surface per minute. Calculation: 4 ml. × 1.56 = 6.2 ml.)

b) No. 158 *gastrointestinal replacement solution* containing 10% carbohydrate: 375 ml. intravenously at rate of 4.7 ml. per minute in one hour and 20 minutes.

(This solution replaced 375 ml. of intestinal secretions lost by suction during the previous 12-hour period.)

TABLE 25

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Acute Appendicitis NAME OR NUMBER Case 11 SEX F AGE 29

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	1) Decreased fluid intake for 36 hours 2) Failure to void for 8 hours. Suggestive of deficit	1) Decreased fluid intake 2) Complaint of thirst Suggestive of excess	1) Decreased food intake (development of fasting ketosis). Suggestive of base bicarbonate deficit (metabolic acidosis)
Physical Examination	1) Dry skin and mucous membranes. Suggestive of deficit	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	1) Hemoglobin 16 Gm. 2) Hematocrit 47% 3) Urine volume 75 ml. 4) Urine specific gravity 1.032. Indicative of deficit	Negative for deficit or excess	1) Acetone present in urine. Indicative of base bicarbonate deficit (metabolic acidosis)
Diagnosis	Moderate fluid volume deficit	Probable electrolyte concentration excess	Base bicarbonate deficit (metabolic acidosis)

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Caloric deficit indicated by cessation of oral intake.

Management

1) Body surface area: 1.56 square meters.
2) First infusion--
    a) No. 51 pump priming solution containing 10% carbohydrate: 250 ml. intravenously at rate of 12.5 ml. per minute in 20 minutes followed by
    b) No. 51 pump priming solution containing 10% carbohydrate: 100 ml. intravenously at rate of 3.1 ml. per minute during 32 minutes required for operative procedure followed by
    c) No. 90 balanced solution containing 10% carbohydrate: 2000 ml. intravenously at rate of 4.7 ml. per minute in seven hours and six minutes.
3) Second infusion, begun approximately 14 hours after completion of operation–
    a) No. 90 balanced solution containing 10% carbohydrate: 1400 ml. intravenously at rate of 4.7 ml. per minute in five hours followed by
    b) No. 158 gastrointestinal replacement solution containing 10% carbohydrate: 450 ml. intravenously at rate of 4.7 ml. per minute in one hour and 36 minutes.

TABLE 26

FLUID IMBALANCE ANALYSIS

Day 2

ADMISSION DIAGNOSIS Acute Appendicitis NAME OR NUMBER Case 11 SEX F AGE 29

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Diagnosis	Normal	Normal	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Normal

Management

1) First infusion--
    - a) No. 90 balanced solution containing 10% carbohydrate: 1200 ml. intravenously at rate of 6.2 ml. per minute in three hours and 14 minutes followed by
    - b) No. 158 gastrointestinal replacement solution containing 10% carbohydrate: 375 ml. intravenously at rate of 4.7 ml. per minute in one hour and 20 minutes.

2) Second infusion, begun 12 hours after start of first infusion--
    - a) No. 90 balanced solution containing 10% carbohydrate: 1200 ml. intravenously at rate of 6.2 ml. per minute in three hours and 14 minutes followed by
    - b) No. 158 gastrointestinal replacement solution containing 10% carbohydrate: 350 ml. intravenously at rate of 4.7 ml. per minute in one hour and 14 minutes.

2) Second infusion, begun 12 hours after start of first infusion—

a) No. 90 *balanced solution* containing 10% carbohydrate: 1200 ml. intravenously at rate of 6.2 ml. per minute in three hours and 14 minutes followed by

(This was the second portion of the calculated daily dose of *balanced solution.*)

b) No. 158 *gastrointestinal replacement solution* containing 10% carbohydrate: 350 ml. intravenously at rate of 4.7 ml. per minute in one hour and 14 minutes.

(This solution replaced intestinal secretions obtained by suction during the previous 12-hour period.)

Comment

This case presents a patient who had sustained a moderate fluid volume deficit, probable electrolyte concentration excess and a base bicarbonate deficit (metabolic acidosis) during the course of an attack of acute appendicitis. These imbalances resulted from inadequate food and fluid intake combined with continuing loss of water from skin and lungs and a fasting ketosis. They were manifested by:

thirst
failure to void for eight hours
dry skin and mucous membranes
probable elevation of hemoglobin and hematocrit
small bladder urine volume
urine specific gravity 1.032
acetonuria

A *pump priming solution* was given initially because of renal depression secondary to the moderate fluid volume deficit. The *balanced solution* corrected the fluid volume deficit. Free water in the *balanced solution* corrected the electrolyte concentration excess. Lactate in the solution supported the base bicarbonate side of the carbonic acid: base bicarbonate ratio. Carbohydrate in both solutions provided calories to combat ketosis.

The postoperative losses of intestinal secretions from intestinal suction were replaced quantitatively by No. 158 *gastrointestinal replacement solution.*

The patient made an uneventful recovery and was discharged from the hospital seven days after admission.

CASE 12. A physician was called to the home of an 80-year-old farmer who had become increasingly disoriented over a two-day period. History revealed that the patient had a carcinoma of the prostate gland, with local metastases, for which he was receiving a synthetic estrogen. He had complained of pain in the right side of his chest for 10 days, and for five days had been confined to bed. The cause of the pain was undetermined but it was gradually subsiding. The patient's appetite had been poor during the 10-day period but he had consumed large amounts of ice water. The outside temperature had been above 95° F. every day for a week, and the patient had perspired profusely during this period. He had voided frequently during the time the disorientation developed. *Physical examination* revealed a confused old man with

oral temperature 99.6° F.
pulse 100
blood pressure 110/70
respirations 18
somewhat sluggish superficial and deep tendon reflexes

Clinical Progress

The attending physician considered the possibility of a cerebral thrombosis or malignant metastasis to the brain. In the absence of any significant findings upon neurological examination, he decided that a therapeutic trial of oral sodium chloride was in order. The patient's disorientation disappeared within 24 hours.

The fluid balance diagnosis and management are summarized in Table 27.

Explanation of Management as Presented in Table 27

1) Sodium chloride as two 0.30 Gm. sodium chloride tablets by mouth every three hours.

(The salt tablets given every three hours furnished, during the 24-hour period, 82 mEq. of sodium and 82 mEq. of chloride, adequate sodium and chloride to correct the deficit.)

Comment

This case illustrates a mild electrolyte concentration deficit of the extracellular fluid. This type of fluid imbalance may occur during hot weather in bed patients whose food intake is poor. Because of poor food intake, sodium chloride intake is low. The kidneys are able to conserve body sodium to the extent that practically none is excreted in the urine. However, sodium chloride is lost in perspiration. An electrolyte concentration deficit of extracellular fluid will gradually develop unless supplemental sodium chloride is administered.

TABLE 27

FLUID IMBALANCE ANALYSIS

Day 1

ADMISSION DIAGNOSIS Disorientation of Unknown Etiology NAME OR NUMBER Case 12 SEX M AGE 80

Properties of Extracellular Fluid

	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History	Negative for deficit or excess	1) Disorientation 2) Poor food intake 3) Profuse perspiration 4) Pronounced water consumption. Suggestive of deficit	Negative for deficit or excess
Physical Examination	Negative for deficit or excess	Negative for deficit or excess	Negative for deficit or excess
Laboratory Findings	None available	None available	None available
Diagnosis	Normal	Mild electrolyte concentration deficit	Normal

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked.

Protein and caloric deficit suggested by poor food intake.

Management 1) Sodium chloride as two 0.30 Gm. sodium chloride tablets by mouth every three hours.

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Workshop

I wish that practising was not so different from preaching.

John Godfrey Saxe

The information presented in this handbook should enable the practitioner to diagnose and treat most of the fluid balance problems encountered in the everyday practice of medicine. This chapter presents six case histories, each of which represents a typical fluid balance problem. A blank *fluid imbalance analysis sheet* accompanies each case history. The reader is urged to test his knowledge of what he has read by recording his fluid balance diagnosis and fluid therapy for each case on the blank analysis sheet. Our fluid balance diagnosis and therapy for each case are presented at the end of the chapter. If diagnosis and therapy for each case are not readily apparent, a rereading of previous pertinent chapters is suggested.

Smooth sailing!

PROBLEMS

CASE 1. A 1½-year-old girl was admitted to the hospital because of vomiting and diarrhea of three days' duration. History revealed the patient's parents and siblings had suffered from similar complaints during the previous week. The patient had passed 10 to 12 loose watery stools a day since onset of the illness. She retained fluids for the first 36 hours of the illness, but vomited or refused all feedings after that period. She had not voided for 16 hours. Her weight at the onset of the illness was estimated to be 25 pounds.

Physical examination revealed a lethargic infant with
- rectal temperature 101° F.
- pulse 115
- respirations 30
- dry skin and mucous membranes
- poor tissue turgor
- increased bowel sounds
- weight 10 Kg. (22 pounds)
- height 32 inches

Laboratory examination of the blood revealed
- hemoglobin 13.5 Gm.
- white blood count 10,000 with 50% polymorphonuclear cells
- plasma bicarbonate 9 mEq./L.
- plasma chloride 105 mEq./L.

Urine specimen not obtained.

Clinical Progress

The patient voided 30 minutes after the start of fluid therapy. She had three loose stools during the first 12 hours with an approximate total volume of 120 ml. No further diarrhea occurred. She did not vomit after admission. Parenteral fluid therapy was not necessary after the first day.

Fluid balance diagnosis?
Fluid therapy?

Fluid Imbalance Analysis

Admission Diagnosis____________________ Name or Number____________ Sex_____ Age_____

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History			
Physical Examination			
Laboratory Findings			
Diagnosis			

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked

Management

CASE 2. A 16-year-old boy was admitted to the hospital because of abdominal pain. History revealed pain of 12 hours duration. It had been associated with nausea from the time of onset. Initially, the pain was in the epigastric area. Later it shifted to the right lower abdomen. The patient had vomited once. Only a few sips of water had been retained. The patient had voided two hours before admission. No stool had been passed for 24 hours.

Physical examination revealed
  oral temperature 100.6° F.
  pulse 105
  respirations 19
  blood pressure 116/76
  dry mucous membranes of the mouth
  good tissue turgor
  point tenderness, rigidity, and rebound tenderness in the right lower quadrant
  weight 68 Kg. (150 pounds)
  height 67 inches

Laboratory examination of the blood revealed
  hemoglobin 15 Gm.
  hematocrit 44%
  white blood count 14,000 with 92% polymorphonuclear cells

Urinalysis revealed
  pH 6.0
  specific gravity 1.028
  otherwise negative

Clinical Progress

A clinical diagnosis of acute appendicitis was made. The inflamed appendix was removed without difficulty. The patient was nauseated during the first postoperative day but did not vomit. Specific fluid therapy was not necessary after the first postoperative day.

Fluid balance diagnosis?

Fluid therapy?

FLUID IMBALANCE ANALYSIS

ADMISSION DIAGNOSIS________________ NAME OR NUMBER__________ SEX____ AGE_____

Properties of Extracellular Fluid

	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History			
Physical Examination			
Laboratory Findings			
Diagnosis			

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked

Management

CASE 3. A 40-year-old female was admitted to the hospital for an elective cholecystectomy because of recurrent cholecystitis and cholelithiasis.
Physical examination revealed
oral temperature 98.6° F.
pulse 76
respirations 16
blood pressure 130/85
moderate obesity
weight 64 Kg. (141 pounds)
height 63 inches
Laboratory examination of the blood revealed
hemoglobin 14 Gm.
hematocrit 42%
white blood count 8500 with a normal differential
Urinalysis revealed
pH 6.5
specific gravity 1.022
otherwise negative

Clinical Progress

The operative procedure was completed in two hours. The patient suffered from considerable nausea and abdominal distention during the first 24 hours after operation. A suction tube was inserted into the duodenum for the first 24 hours postoperatively; 300 ml. of intestinal secretion was removed during each 12-hour period.

Fluid balance diagnosis?

Fluid therapy?

FLUID IMBALANCE ANALYSIS

ADMISSION DIAGNOSIS________________ NAME OR NUMBER____________ SEX____ AGE____

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History			
Physical Examination			
Laboratory Findings			
Diagnosis			

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked

Management

CASE 4. A 25-year-old male was admitted to the hospital because of increasing drowsiness of six hours duration. He was known to be diabetic. History revealed carelessness in administration of insulin. He had had no insulin for two days, although his prescribed dose was 55 units per day. Fluid intake during the 18 hours before admission consisted of a few sips of water. During the 12 hours prior to admission, he had vomited a number of times. He had continued to void frequently despite the poor fluid intake.

Physical examination revealed a semicomatose man with

rectal temperature 101° F.
pulse 100
respirations 25 (deep and regular)
blood pressure 120/80
dry skin and mucous membranes
poor tissue turgor
acetone odor to the breath
estimated weight 69 Kg. (152 pounds)
height 67 inches

Laboratory examination of the blood revealed

hemoglobin 17 Gm.
hematocrit 50%
white blood count 16,000 with 65% polymorphonuclear cells
blood sugar 300 mg./100 ml.
plasma bicarbonate 6 mEq./L.
plasma chloride 100 mEq./L.

Urinalysis revealed

pH 5.6
specific gravity 1.028
4 plus sugar
acetone present
diacetic acid present

Clinical Progress

Large doses of insulin were given along with parenteral fluid therapy. An estimated 800 ml. of fluid was lost during the first 12 hours of treatment because of vomiting, perspiration and hyperventilation (increased insensible water loss). The patient began to tolerate oral fluids 14 hours after start of therapy. Intravenous fluids were discontinued about 17 hours after start of therapy.

Fluid balance diagnosis?

Fluid therapy?

FLUID IMBALANCE ANALYSIS

ADMISSION DIAGNOSIS________________ NAME OR NUMBER __________ SEX____ AGE____

Properties of Extracellular Fluid

	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History			
Physical Examination			
Laboratory Findings			
Diagnosis			

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked

Management

CASE 5. A 31-year-old male was admitted to the hospital because of severe epigastric pain. History revealed that the patient had suffered from a duodenal ulcer intermittently for the past five years. Two days prior to admission, the patient suffered a recurrence of ulcer symptoms. Two hours before admission, the patient experienced severe epigastric pain.

Physical examination revealed a young man in severe pain with
- oral temperature 98° F.
- pulse 88
- respirations 22
- blood pressure 110/75
- tenderness and board-like rigidity of the upper abdomen
- absent bowel sounds
- weight 65 Kg. (143 pounds)
- height 70 inches

Laboratory examination of the blood revealed
- hemoglobin 15 Gm.
- hematocrit 44%
- white blood count 15,000 with 75% polymorphonuclear cells

Urinalysis revealed
- pH 6.0
- specific gravity 1.014
- otherwise negative

Roentgenogram of the abdomen in the erect position revealed free air beneath the diaphragm.

Clinical Progress

The patient had a ruptured duodenal ulcer which was repaired one hour after admission to the hospital. Gastric suction was necessary for 24 hours; 450 ml. of fluid was removed during each 12-hour period after operation. This fluid had an average pH of 3.0.

Fluid balance diagnosis?

Fluid therapy?

FLUID IMBALANCE ANALYSIS

ADMISSION DIAGNOSIS________________ NAME OR NUMBER____________ SEX_____ AGE_____

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History			
Physical Examination			
Laboratory Findings			
Diagnosis			

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked

Management

CASE 6. An 18-year-old male was admitted to the hospital in a state of collapse. History revealed that he collapsed after four hours of football practice. The outside temperature at the time was above 90° F. The patient perspired profusely during the entire practice session. His teammates stated he drank at least three or four quarts of water during the four-hour period.

Physical examination revealed a pale, semicomatose young man with
- oral temperature 99° F.
- pulse 120
- respirations 18
- blood pressure 85/50
- weight 75 Kg. (165 pounds)
- height 70 inches

Laboratory examination of the blood revealed
- hemoblogin 14 Gm.
- hematocrit 40%
- white blood count 10,000 with normal differential
- plasma sodium 130 mEq./L.

Urinalysis revealed
- pH 6.0
- specific gravity 1.002
- otherwise negative

Clinical Progress

Redetermination of patient's plasma sodium after one-half of the calculated parenteral fluid dose had been infused was 135 mEq./L. The patient responded well to treatment and was discharged from the hospital the following day.

Fluid balance diagnosis?

Fluid therapy?

FLUID IMBALANCE ANALYSIS

ADMISSION DIAGNOSIS________________ NAME OR NUMBER__________ SEX_____ AGE_____

	Properties of Extracellular Fluid		
	Volume	*Total Electrolyte Concentration*	*Electrolyte Composition*
Clinical History			
Physical Examination			
Laboratory Findings			
Diagnosis			

Position of extracellular fluid, and nutritional status of patient as to protein, calories and vitamins not listed but should be checked

Management

ANSWERS

CASE 1.

Fluid balance diagnosis:

1) Severe fluid volume deficit.
2) Base bicarbonate deficit (metabolic acidosis).
3) Probable potassium deficit.

Fluid therapy:

1) Body surface area: 0.48 square meters.
2) Single infusion—

a) No. 51 *pump priming solution* containing 10% carbohydrate: 114 ml. intravenously at rate of 3.8 ml. per minute in 30 minutes followed by

b) No. 75 *balanced solution* containing 10% carbohydrate: 663 ml. intravenously at rate of 0.96 ml. per minute in 11 hours and 30 minutes followed by

c) No. 75 *balanced solution* containing 10% carbohydrate: 783 ml. intravenously at rate of 1.1 ml. per minute during the next 12 hours.

Comment

The quantity of *pump priming solution* given was subtracted from the dose of *balanced solution* calculated to provide water and electrolytes for maintenance and to correct the severe fluid volume deficit. The concurrent loss of approximately 120 ml. in diarrheal stools was corrected by the addition of 120 ml. of *balanced solution* to the *balanced solution* given during the second 12 hours.

CASE 2.

Fluid balance diagnosis:

No fluid imbalance present.

Fluid therapy:

1) Body surface area: 1.79 square meters.

2) First infusion, begun after operation—No. 90 *balanced solution* containing 10% carbohydrate: 1350 ml. intravenously at rate of 7.2 ml. per minute in three hours and eight minutes.

3) Second infusion, begun 12 hours after start of first infusion—No. 90 *balanced solution* containing 10% carbohydrate: 1350 ml. intravenously at rate of 7.2 ml. per minute in three hours and eight minutes.

CASE 3.

Fluid balance diagnosis:

No fluid imbalance present.

Fluid therapy:

1) Body surface area: 1.66 square meters.

2) First infusion, begun after operation—No. 75 *balanced solution* containing 10% carbohydrate: 1250 ml. intravenously at rate of 6.6 ml. per minute in three hours and 10 minutes.

3) Second infusion, begun 12 hours after completion of operation—

a) No. 75 *balanced solution* containing 10% carbohydrate: 1250 ml. intravenously at rate of 6.6 ml. per minute in three hours and 10 minutes followed by

b) No. 158 *gastrointestinal replacement solution* containing 10% carbohydrate: 300 ml. intravenously at rate of 5 ml. per minute in one hour.

Comment

On the second postoperative day the patient was given oral *balanced solution* ad libitum. She drank and retained approximately 3000 ml. which provided water and electrolytes for maintenance and for replacement of the concurrent loss of the previous 12 hours.

CASE 4.

Fluid balance diagnosis:

1) severe fluid volume deficit
2) base bicarbonate deficit (metabolic acidosis)
3) probable potassium deficit

Fluid therapy:

1) Body surface area: 1.80 square meters.

2) Single infusion—No. 75 *balanced solution* containing 10% carbohydrate: 5400 ml. intravenously at rate of 5.4 ml. per minute in 16 hours and 38 minutes.

3) Oral *balanced solution* given ad libitum, begun 14 hours after start of parenteral fluid therapy: 1000 ml. taken and retained during the next 10 hours.

CASE 5.

Fluid balance diagnosis:

No fluid imbalance present

TABLE 28

COMMERCIALLY AVAILABLE SOLUTIONS CORRESPONDING TO SOLUTIONS DESCRIBED IN THIS HANDBOOK

Generic Name in Handbook			*Abbott Laboratories*	*Baxter Laboratories Inc.*	*Cutter Laboratories*	*Mead Johnson & Company*	*Generic Name in Handbook*		
PARENTERAL	Pump priming solutions	No. 51	Dextrose 5% in Quarter Strength Saline	5% Dextrose in 0.2% Sodium Chloride	Dextrose 5% in ½ NSS	Dextrose 5% in 0.33% Sodium Chloride	No. 51	Pump priming solutions	PARENTERAL
		No. 70	——	——	——	——	No. 70		
	Balanced solutions	No. 48	——	Travert® 5% with Polyionic No. 1	——	Dextrose 5% Levugen® 5% or Levugen® 10% with Electrolyte No. 48	No. 48	Balanced solutions	
		No. 75	——		——	Dextrose 5% or Levugen® 5% with Electrolyte No. 75	No. 75		
		No. 90	Inonsol® B with Dextrose 5%	Travert® 10% Electrolyte No. 2	Invert Sugar 10% in Electrolyte No. 2	Levugen® 10% with Electrolyte No. 90	No. 90		
	Gastro-intestinal replacement	No. 130	——	1) Modified Duodenal Solution 2) Travert® 10% —Electrolyte No. 1	Invert Sugar 10% in Electrolyte Solution No. 1	——	No. 130	Gastro-intestinal replacement	
		No. 150	Ionosol® G with Dextrose 10% or Invert Sugar 10%	Travert® 10% Electrolyte No. 3	Invert Sugar 10% in Electrolyte Solution No. 3	Levugen® 10% with Electrolyte No. 150.	No. 150		

SOLUTIONS	solutions	No. 158	Ionosol® D with Dextrose 10% or Invert Sugar 10%	———	Polysal® with 5% Dextrose	Levugen® 5% or Levugen® 10% with Electrolyte No. 158	No. 158	solutions	SOLUTIONS
		Lactated Ringer's solution	1) Lactated Ringer's Solution 2) Dextrose 5% or 10% in Lactated Ringer's Solution	1) Lactate Ringer's 2) 5% or 10% Dextrose in Lactate Ringer's	1) Hartmann's Solution 2) Dextrose 2½%, 5% or 10% in Hartmann's Solution	1) Lactated Ringer's Solution 2) Dextrose 2½%, 5% or 10% in Lactated Ringer's Solution	Lactated Ringer's solution		
	Sodium Chloride solutions	Isotonic solution of Sodium chloride	Isotonic Sodium Chloride Solution (also available with Dextrose 2½%, 5%, 10% and Invert Sugar 5% and 10%)	Normal Saline (also available with Dextrose 2½%, 5%, 10% and Travert® 5% and 10%)	Normal Saline (also available with Dextrose 2½%, 5%, 10% and Invert Sugar 5% and 10%)	Normal Saline (also available with Dextrose 2½%, 5%, 10% Levugen® 10% and Invert Sugar 10%)	Isotonic solution of Sodium chloride	Sodium Chloride solutions	
		3% Sodium chloride	———	3% Sodium Chloride	———	3% Sodium Chloride	3% Sodium chloride		
		5% Sodium chloride	Sodium Chloride 5% Solution	5% Sodium Chloride	5% Sodium Chloride	———	5% Sodium chloride		
	Protein hydrolysate 5% in 5% carbohydrate solution with electrolyte content similar to balanced solutions	No alcohol	Aminosol® 5% with Dextrose 5% and Sodium Chloride 0.3% Solution	———	CPH Solution (Amino Acid 5%, Dextrose 5%) in Water	Amigen® 5%, Dextrose 5%	No alcohol	Protein hydrolysate 5% in 5% carbohydrate solution with electrolyte content similar to balanced solutions	
		With alcohol	Aminosol® 5% with Dextrose 5% in Alcohol 5%	———	———	Amigen® 5%, Dextrose 5%, Alcohol 5%	With alcohol		

TABLE 28

Commercially Available Solutions Corresponding to Solutions Described in This Handbook

	Generic Name in Handbook		*Abbott Laboratories*	*Baxter Laboratories Inc.*	*Cutter Laboratories*	*Mead Johnson & Company*	*Generic Name in Handbook*		
PARENTERAL SOLUTIONS	Carbohydrate in water solutions		1) Dextrose 2½%, 5% and 10% in Water 2) Invert Sugar 5% and 10% in Water 3) Fructose 10% in Water	1) Dextrose 2½%, 5% and 10% in Water 2) Levulose 10% in Water 3) Travert® 5% and 10% in in Water	1) Dextrose 2½%, 5% and 10% in Water 2) Invert Sugar 5% and 10% in Water 3) Levulose 10% in Water	1) Dextrose 2½%, 5% and 10% in Water 2) Levugen® 5% and 10% in Water 3) Invert Sugar 10% in Water	Carbohydrate in water solutions		PARENTERAL SOLUTIONS
	Other solutions	⅙ Molar sodium lactate solution	Sodium r-Lactate ⅙ Molar Solution	One-sixth Molar Sodium r-Lactate Solution	M/6 Sodium Lactate Solution	M/6 Sodium Lactate Solution	⅙ Molar sodium lactate solution	Other solutions	
		Ammonium chloride solution	Ammonium Chloride 400 Milliequivalents Solution	2.14% Ammonium Chloride in Water	2.14% Ammonium Chloride in Water	——	Ammonium chloride solution		
		Ammonium chloride concentrate	Ionotrate® Ammonium Chloride Solution—100 mEq. in 20 ml.	——	——	Ammonium Chloride Concentrate 50 mEq. in 50 ml.	Ammonium chloride concentrate		
		Dextran	Dextran 6% W/V in Saline	Gentran®	Dextran 6% Injection/Cutter in Normal Saline	Plasran® (Dextran 6% Mead)	Dextran		
ORAL SOLUTIONS	Oral balanced solution		——	——	——	Lytren®	Oral balanced solution		ORAL SOLUTIONS
	High protein high calorie high vitamin nutritional mixture		——	——	——	Sustagen®	High protein high calorie high vitamin nutritional mixture		

Fluid therapy:

1) Body surface area: 1.80 square meters.

2) First infusion, begun after operation—No. 90 *balanced solution* containing 10% carbohydrate: 1350 ml. intravenously at rate of 7.2 ml. per minute in three hours and six minutes.

3) Second infusion, begun 12 hours after completion of operation—

a) No. 90 *balanced solution* containing 10% carbohydrate: 1350 ml. intravenously at rate of 7.2 ml. per minute in three hours and six minutes followed by

b) No. 150 *gastrointestinal replacement solution* containing 10% carbohydrate: 450 ml. intravenously at rate of 5.4 ml. per minute in one hour and 23 minutes.

Comment

On the second postoperative day the patient was given two infusions of the *balanced solution* at the maintenance level of 1500 ml. per square meter of body surface per day. The *gastrointestinal replacement solution* was given after the first infusion to replace the concurrent loss from gastric suction of the previous 12 hours. On the third postoperative day the patient's maintenance requirement for water and electrolytes was met by ad libitum administration of oral *balanced solution*.

CASE 6.

Fluid balance diagnosis:

Electrolyte concentration deficit of extracellular fluid

Fluid therapy:

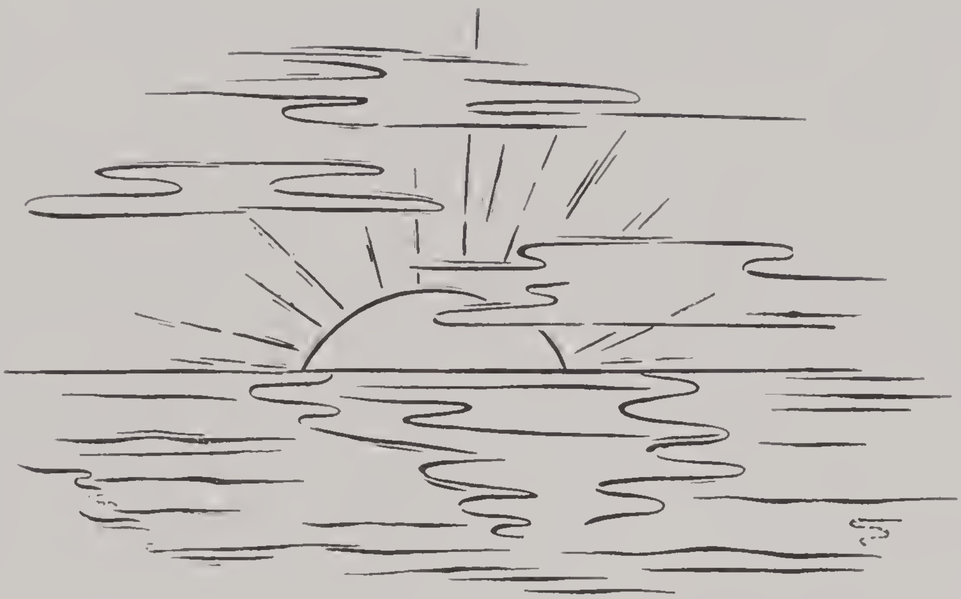
1) Body surface area: 1.92 square meters

2) First infusion—3% sodium chloride solution: 132 ml. intravenously at rate of 1.9 ml. per minute in one hour and 10 minutes

3) Redetermination of plasma sodium: 135 mEq./L.

4) Second infusion—3% sodium chloride solution: 132 ml. intravenously at rate of 1.9 ml. per minute in one hour and 10 minutes.

Epilogue



The sea has always fascinated men—awing them with its beauty, its thundering might, its limitless expanse and its majesty. Often it has kindled the desire to learn what lay beyond the distant horizon. Never has this effect of the all-encircling sea been better expressed than in a song of those ancient navigators and seafarers, the Polynesians:

> The sun sags down on Tama's path,
>   across the changing sky;
> New stars do leap above the deep
>   to meet the wondering eye;
> New seas are spread on every side;
>   New skies are overhead;
> New lands await the sea-kings;
>   in the vast grey seas ahead.
>
> MAORI SONG[*]

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[*] George Grady Press: selection from "Maori Song" in *The Raft Book* by Harold Gatty; used by permission of Harold Gatty and Mrs. Harold Gatty, copyright owners.

The body fluids are man's heritage from the sea. Our understanding of them is truly in its infancy. Consider: A scant twenty years ago we knew virtually nothing of the clinical importance of the potassium ion! Medical science has barely penetrated the surface of knowledge of the body fluids; the depths remain to be plumbed. Surely countless exciting discoveries await the physician, investigator and practitioner alike, in the vast grey realm of our internal seas, the body fluids.

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This Book

FLUID BALANCE HANDBOOK FOR PRACTITIONERS

By

WILLIAM D. SNIVELY, JR., M.D.

AND

MICHAEL J. SWEENEY, M.D.

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Anions

Conversion Table: Milligrams per hundred milliliters to milliequivalents per liter

CHLORIDE	
mg./100 ml.	*mEq./L.*
240	67
260	73
280	79
300	84
320	90
340	96
360	101
380	107
400	113
420	118
440	124

To convert mg./100 ml. to mEq./L. multiply by 0.282

CHLORIDE AS SODIUM CHLORIDE	
mg./100 ml.	*mEq./L.*
430	73
460	78
490	84
520	89
550	94
580	99
610	104
640	109
670	114
700	120
730	125

To convert mg./100 ml. to mEq./L. multiply by 0.171

PHOSPHORUS	
mg./100 ml.	*mEq./L.*
1	0.58
2	1.16
3	1.74
4	2.32
5	2.90
6	3.48
7	4.06
8	4.64
9	5.22
10	5.80

To convert mg./100 ml. to mEq./L. multiply by 0.580

BICARBONATE	
CO_2 Content or CO_2–Combining Power Volumes per 100 ml.	*mEq./L.*
10	4.5
20	9.0
30	13.5
40	18.0
50	22.5
60	27.0
70	31.5
80	36.0
90	40.5
100	45.0
110	49.5

To convert volumes per 100 ml. to mEq./L. multiply by 0.45

CFTRI-MYSORE
4004
Fluid balance ha

Cations

Conversion Table: Milligrams per hundred milliliters to milliequivalents per liter

SODIUM	
mg./100 ml.	*mEq./L.*
270	117
280	122
290	126
300	130
310	135
320	139
330	143
340	148
350	152
360	156
370	160

To convert mg./100 ml. to

POTASSIUM	
mg./100 ml.	*mEq./L.*
5	1.28
10	2.56
15	3.84
20	5.12
25	6.40
30	7.68
35	8.96
40	10.24

To convert mg./100 ml. to mEq./L. multiply by 0.256

CALCIUM	
g./100 ml.	*mEq./L.*
4	2.0
5	2.5
6	3.0
7	3.5
8	4.0
9	4.5
10	5.0
11	5.5
12	6.0
13	6.5
14	7.0

o convert mg./100 ml. to Eq./L. multiply by 0.5

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